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P. J. DU TOIT,
Director of Veterinary Services.

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15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

Haemo-Lymphoid-like Nodules in the Liver of Ruminants a few years after Splenectomy.

By G. DE KOCK, M.R.C.V.S., Dr.Med.Vet., D.Sc., Sub-Director of Veterinary Services.

The pathology of splenectomy in clinically healthy sheep, and in sheep affected with anaplasmosis, was studied by De Kock (1928). He came to the following conclusions:—

(1) That the reticulo-endothelial system of clinically healthy sheep is so developed that it can, after the removal of the spleen, deal with normal blood destruction, without manifesting any specific alterations;

(2) that extensive erythrophagocytosis and desquamation of stern cells in the liver were observed in cases of splenectomized sheep affected with anaplasmosis. The liberation of the stern cells was reflected in the circulation as a monocytosis.

All these observations were made in sheep that had died or were killed at intervals within 15 months after splenectomy.

Since then interesting changes were detected in the livers of a number of ruminants that had died or were killed about three years after splenectomy. All these animals, without exception, showed the presence of multiple nodules in the liver. At first it was thought that they were of the nature of a neoplasma, but the microscopical examination, and the regular occurrence of these nodules after splenectomy, substantiated the fact that they were new tissue formations. In some instances they resembled the structure of haemolymph glands very closely, and for time being they have been designated as haemolymphoid-like nodules.

The object of this paper is to describe and discuss the nature of these peculiar nodules seen in the livers of splenectomized ruminants. A brief consideration of the literature will be undertaken to ascertain to what extent similar nodules have been described in man and other animals.

In Table No. 1 an attempt has been made to enumerate very briefly the following data in respect of all animals splenectomized at Onderstepoort:—

Column (i): the ear number of the animal splenectomized;
Column (ii): previous history, if any;
Column (iii): age of the animal at the time of splenectomy;
Column (iv): date of splenectomy;
Column (v): result of splenectomy;
Column (vi): subsequent history;
Column (vii): the number of the specimen collected at post-mortem for microscopical examination.

TABLE 1.

No. of Animal.	Previous History.	Age.	Splenectomized.	Result.	Subsequent History.	Specimen No.
Equines.—						
15186	H.S. Expt.	2½ years	25/10/23	Rel. N. 29/10/23	Killed *in extremis* 31/10/23	3200
15420	—	5 years	21/11/23	Rel. N. 22/11/23	Died N. 24/11/23	3313
16072	—	3 months	7/3/24	—	Died haemolysis expt. 15/6/24	4108
16032	—	11 months	23/10/24	Rel. N. 29/10/24	Killed *in extremis* 31/10/24	4478
Bovines.—						
711	—	1 year	27/3/24	Rel. N. 19/5/24	T.P. 6/3/25, P. 10/3/25, Died P. 14/3/25	4813
758	—	9 months	6/6/24	—	Died 26/6/24, paratyphoid and bleeding	4122
828	—	10 months	20/11/24	—	Died 23/11/24, H.W. (?)	4512
893	—	9 months	15/12/24	Rel. P. + A.	E.C.F., Tryp. expts. Died 7/10/27, Tryp.	N.B. 7408
1027	—	2 months	24/12/24	Rel. P. + A.	E.C.F. expts. Died P. + E.C.F. 22/5/25	4966
1034	—	3 months	20/1/25	Rel. P. + A. 10/2/25	T. P. + A. Died P. 5/6/28	N.B. 8186
870	E.C.F. Expt.	—	30/4/25	Rel. A.	2/4/28 Kaalplaats. Died I., P. 3/5/28	N.B. 8016
894	E.C.F. Expt.	1 year	17/3/25	Rel. A.	2/4/28, Kaalplaats. Died I., P. 21/5/28	N.B. 8055
1032	—	—	21/4/25	Rel. P. + A.	10/2/28, West Camp. Died Rei. P. 20/3/28	N.B. 7852
Goats.—						
8280	—	18 months	30/5/24	—	I.A. 7/12/24	—
8304	—	18 months	30/5/24	—	I.A. 24/11/24, killed 19/7/28	N.B. 8230
Sheep.—						
7443	—	Full-mouth	29/2/24	Rel. A. 13/3/24	Died A. 21/3/24	3859
7369	—	Full-mouth	29/2/24	Rel. A. 13/3/24	Killed *in extremis* 19/3/24	3844
8430	—	Lamb	8/4/24	—	T.A. 15/9/24, A. 22/8/24, killed 30/9/24	4444
8431	—	Lamb	8/4/24	Died 12/4/24 (?)		3898
8456	—	Lamb	15/5/24	—	T.A. 26/6/24, A. 30/6/24, killed 11/7/24 for path.	4143
8457	—	Lamb	15/5/24	—	T.A. 26/6/24, A. 7/7/24. Killed 16/9/24 for pathology.	4349

TABLE 1—(*continued*).

No. of Animal.	Previous History.	Age.	Splenectomized.	Result.	Subsequent History.	Specimen No.
Sheep.—						
8464	—	Lamb	15/5/24	—	Died H.W. 11/9/24	—
8427	31/3/24 A.	Lamb	3/7/24	Rel. A. 21/7/24	Killed 18/11/25	5592
8428	2/4/24 A.	Lamb	3/7/24	Rel. A. 11/7/24	Killed 19/7/28	N.B. 8231
8429	30/3/24 A.	Lamb	22/9/24	Rel. A. 13/10/24	—	—
8434	27/3/24 A.	Lamb	3/7/24	Rel. A. 11/7/24	Died A. 19/7/24	4175
8462	30/5/24 A.	Lamb	22/9/24	Splenic vessels ligatured. Died 25/9/24	—	—
8455	5/5/24 A.	Lamb	16/1/25	Died ?, complicated with previous anaemia	—	4645
8458	24/4/24 A.	Lamb	16/1/24	Died ?, 21/1/25, complicated with previous anaemia	—	4663
8451	22/10/24 A.	—	28/1/25	Rel. A. 9/2/25	Died 8/4/27, anaemia	6750
9119	7/10/24 A.	—	28/1/25	Rel. A. 28/2/25	Died, 20/5/26, bact. icterus	5949
10511	21/1/25 A.	—	12/3/25	Rel. A. 30/3/25	Died 7/11/27, uraemia	7494
9414	—	—	6/11/25	—	Killed for path. 11/11/25	5575
10946	—	—	11/5/25	—	Died pneumonia 14/5/25	5591
10656	—	—	6/11/25	Rel. A. 16/11/25	Killed for path. 11/11/25	4945
13853	—	—	21/5/26	Rel. A.	8/6/26, died anaesthesia + A.	5978
10944	—	—	11/5/25	Rel. A. 16/7/25	Died 21/2/28, tar expt	M. 7765
10743	—	—	12/3/25	Rel. A. 1/4/25	—	—
16023	—	—	12/4/27	—	I.A. 29/4/27, A. 12/5/27	—

ABBREVIATIONS.

P. = Piroplasmosis.
A. = Anaplasmosis.
N. = Nuttalliosis.
Rel. = Relapse.
Rei. = Reinfection.
H.S. = Horsesickness.
H.W. = Heartwater.

T. = Tested.
M. = Metaplasia.
I. = Injected.
E.C.F. = East Coast Fever.
H.W. = Heartwater.
Tryp. = Trypanosomiasis.

The pathology of the majority of cases referred to in the above table will be found in the publication of De Kock. No specific alterations were recorded by him in any of the cases which died or were killed prior to 1928.

These characteristic nodules were for the first time seen in the following splenectomized bovines which died of piroplasmosis, etc., as a result of artificial or natural infections:—

No. of Bovine.	When Splenectomized.	Date of Death.	No. of Specimen.
893	15/12/24	7/10/27	7408
1032	21/4/25	20/3/28	7852
870	30/4/25	3/5/28	8016
894	17/3/25	21/5/28	8055
1034	20/1/25	5/6/28	8186

The question of this breakdown in immunity is fully discussed by De Kock (1929).

As soon as the writer encountered these changes in the livers of the above-mentioned animals, it was thought advisable carefully to re-examine the livers of all splenectomized animals that had died previously. The object of this re-examination was to see whether nodules or foci had not perhaps been overlooked, or whether earlier stages of this tissue formation in the liver could be detected. The following specimens from bovines were carefully scrutinized without finding any evidence of such changes:—

No. of Specimen.	No. of Bovine.	When Splenectomized.	Date of Death.
4122	758	6/6/24	26/6/24
4512	828	20/11/24	23/11/24
4813	711	27/3/24	14/3/25
4966	1027	24/12/24	22/5/25

The same procedure was adopted in case of all specimens collected from splenectomized sheep, but in all these cases no alterations were detected in any of the earlier cases.

From Table 1 it therefore becomes fully apparent that these tissue formations in the liver did not make their appearance in the liver within a year after splenectomy, nor were these formations seen in the following sheep:—

8427, which died 16 months after splenectomy.
9119, which died 16 months after splenectomy.
8451, which died 26 months after splenectomy.
10511, which died 32 months after splenectomy.

In sheep 10944, which died 33 months after splenectomy, a few definite, but small, nodules were detected in the liver. They were undoubtedly missed at the first examination, probably due to their size and scarcity.

Although the writer was convinced that the diseases from which the five bovines succumbed (i.e. those animals which showed the nodules in the liver) were not in any way responsible for the formation of the nodules, yet it was deemed advisable to compare these findings with those seen in the livers of some of the clinically healthy sheep and goats splenectomized in 1924.

Two of the following splenectomized sheep and goats still under observation were selected and killed on 19th July, 1928, for post-mortem examination:—

No. of Animal.	When Splenectomized.	Specimen No.
Goat—		
8280	30/5/24	[Still alive, 1/3/29.]
8304	30/5/25	8230.
Sheep—		
8428	3/7/24	8231.
8429	22/9/24	[Still alive, 1/3/29.]
10743	12/3/25	[,, ,,]
16023	12/4/27	[,, ,,]

The livers of these two animals, viz., goat 8304 and sheep 8428, revealed similar nodules to those encountered in the bovines. In case of goat 8304 it would appear that these formations did not stand in any relation to piroplasmosis or anaplasmosis.

A complete description of the post-mortem and microscopical appearances, together with photographs, etc., will be found in the Appendix. A description and discussion of the important findings will be dealt wih in the text.

POST-MORTEM CHANGES IN BOVINES.

General anaemia and icterus, haemoglobinaemia, and haemoglobinuria were seen in the majority of cases, as a result of the marked haemolysis produced by *Piroplasma bigeminum*. The majority of animals revealed venous hyperaemia and degenerative changes in the parenchymatous organs. Hydrothorax and ascites were noted in the case which died as a result of trypanosomiasis. Hyperplasia of lymphoid tissue and hypertrophy were noted.

The nodules seen in the liver varied in size from ¼ inch in diameter to about 1½ inches, and even larger (see figures). They were completely circumscribed, and in some instances a capsule could be identified. These nodules showed in all instances a very characteristic dark-red colour, and could be easily identified in virtue of its contrast with the yellow-brown liver substance. These red masses had an outward appearance, somewhat resembling flesh, whereas some showed a mulberry-like appearance on the surface. Small lymphoid follicles could sometimes be clearly identified in these nodules as greyish-white, almost transparent masses. These nodules were raised above the cut surface of the liver, in some instances protruding as much as $\frac{1}{4}$ inch. Some of these nodules could be enucleated from cavities, leaving sacculated bloodvessel-like formations, which were perfectly smooth and glistening. In view of this appearance, it was at first thought that these lesions were of the nature of a thrombosis or an angioma. It was, however, found that these cavities were not in communication with the lumen of any blood-vessel. In some instances this complete enucleation of the nodule was not possible. This was shown by microscopical examination to be due to the fact that these masses in places penetrated the liver substance without any line of demarcation.

These nodules become apparent through the capsule of the liver as dark purple-red circumscribed areas, slightly raised above the surface of the liver. It may be stated that the contour, size, and shape of the livers examined revealed practically no changes.

MICROSCOPICAL APPEARANCES.

It was shown that these masses were made up of large numbers of erythrocytes which were contained in a system of blood-vessels or sinuses (see figures). Associated with the vessel walls were cells, resembling those associated with a haemosiderosis in the spleen. These cells were in a number of instances involved in a pigment metabolism. Some of this pigment was of the nature of a haemosiderosis In some instances the haemosiderosis in the nodule was prominent and extensive, whereas only a limited amount of this pigment formation could be identified in the liver substance. This system of blood-vessels, not arranged according to any particular system or plan, was supported by a delicate connective tissue stroma, which was in the majority of cases continuous with the capsule surrounding the nodule. This capsule in some instances revealed the presence of elastic fibres, circularly arranged around the nodule, in this respect resembling those seen in the capsule of haemo-lymph glands. As pointed out above, the capsule in places shows a break in the continuity, so that the substance of the nodule penetrates the substance of the liver without any line of demarcation. The capsule on the inside seems to be lined by endothelium, which was continuous with the blood-vessels in the nodule. That would explain why the greater portion of the inside of the capsule was perfectly smooth and glistening, resembling the lumina of blood-vessels macroscopically.

Here and there accumulations of round cells could be identified in the nodule, and in some cases these were collected into masses resembling follicles. The round cells were also collected in the form of strands.

The liver substance in the majority of cases showed a venous hyperaemia and fairly extensive fatty changes. In the majority of cases the liver lobule towards the periphery showed a distinct bile stasis.

In the splenectomized clinically healthy sheep the nodules revealed well-formed lymphoid follicles, which showed a well-defined so-called germinal centre (see figures). The cells associated with the walls of the blood vascular system showed very extensive haemosiderosis, and with the Giemsa stain engulfed erythrocyter in all stages of disintegration could be identified in these reticulo-endothelial cells. It may be mentioned here that these nodules in the liver of sheep varied in size from a pin's head to one centimetre in diameter. They contained one or more lymphoid follicles according to the size of the nodule.

The *bone marrow* in sheep 8428 revealed numerous lymphoid follicles. Numerous megakaryocytes were present. Except for these and the presence of the precursors of the blood-cells, the whole structure of the bone marrow resembled haemo-lymphoid-like tissue to a certain extent.

HAEMATOLOGY.

In view of the unexpected deaths in the majority of bovines, it was only possible to make a study of the blood in a few instances. Bovine 1034, i.e. on the day of death, gave the following data as regards the study of its blood:—

Red count: 1.3 millions;
white count: 20,000;
differential count: 48 lymphocytes,
13 monocytes,
39 neutrophiles.

The examination of the bleod-smear revealed the presence of piroplasms in about 90 per cent. of the erythrocytes. In some erythrocytes more than two parasites could be identified. Smears were also made from the following tissues and organs, and stained with May-Grünewald, in order to study the different types of cells present:—

(1) haemo-lymph gland in the subcutaneous tissues;
(2) haemo-lymph gland in peritoneal cavity;
(3) haemo-lymph gland in thoracic cavity;
(4) bronchial lymph gland;
(5) nodules in the liver;
(6) liver substance;
(7) periportal lymph gland;
(8) bone marrow.

Smears Nos. 1, 2, *and* 3 revealed about 90 per cent. of the erythrocytes infected with piroplasms. A number of reticulo-endothelials (?) were detected, showing erythrophagocytoses.

Smear No. 4, i.e. from the bronchial lymph gland, showed a very extensive monocytosis, with erythrophagocytosis. Fully 70 per cent. of all cells were of this type.

Smear No. 5, i.e. from the nodule in the liver, showed a number of reticulo-endothelials with erythrophagocytosis, besides numerous cells of the lymphoid series. Neutrophiles were not infrequent. These smears revealed a fair number of erythrocytes, of which 100 per cent. in places showed the presence of piroplasms. Elongated tissue cells (fibrocytes) were also present. This smear resembled almost in every detail those made from the haemo-lymph glands.

Smear No. 6 revealed liver cells with parts of bile canaliculi filled with bile pigment. Reticulo-endothelials with erythrophagocytosis also observed.

Smear No. 7 showed the typical types of lymphoid cells usually encountered in a lymph-gland smear. Not many erythrocytes were detected.

The bone-marrow smear, i.e. *smear No.* 8, revealed the precursors of leucocytes (the eosinophiles predominating) and erythrocytes. Reticulo-endothelials with erythrophagocytosis were also detected.

Organ smears of goat 8304 were made from the following tissues:—

(1) the liver nodules;
(2) bronchial lymph glands;
(3) haemo-lymph glands.

The smears from the liver nodules again resembled those made from the haemo-lymph glands; both could easily be distinguished from the ordinary type of lymph gland, i.e. *smear No.* 2.

In the case of sheep 8428, organ smears were studied from the following tissues:—

(1) liver nodule;
(2) mediastinal lymph gland;
(3) haemo-lymph gland.

More or less similar findings to those in goat 8304 were obtained.

This question of cell study by means of tissue smears in various organs is to be reported more fully later on. Smears from the spleen of non-splenectomized animals will be included for a complete comparison.

DISCUSSION.

From the above considerations, it would appear that these nodules in the livers of splenectomized ruminants were of the nature of a well-formed tissue and not a type of neoplasm. The tissue to which it had a very close resemblance was the haemo-lymph gland, especially in case of the nodules seen in sheep. These latter revealed well-formed follicles with so-called germinal centres, and even the organ smears had a very close resemblance. They were not of the nature of ordinary lymph follicles. According to Ellenberger and Trautmann (1921), red lymph glands, or blood lymph glands, or haemo-lymph glands, can be differentiated from ordinary lymph glands by the absence of vasa afferentia and efferentia, the presence of wide sinuses filled with blood, weaker capsule, weaker trabeculae, partly absent, etc. According to Jordan (1922), haemo-lymph nodes (haemal glands) are larger and more numerous in the ruminant than in man . . . the lymphoid tissue is enclosed in a capsule, beneath which is a broad sinus filled with blood . . . the peripheral blood sinus, which is analogous to the peripheral lymph sinus of a lymph node, sends into the interior of the organ a greater or less number of secondary sinuses. Schäfer (1916) maintains that the blood passes into these sinuses of the haemo-lymph gland from arterial capillaries . . . like the spleen, these haemal glands show numerous large phagocytes which contain red blood corpuscles in various stages of transformation into pigment . . . some haemal glands are said to have *no* lymph channels, but to be purely blood glands . . . in that case they may be considered to represent accessory spleens . . . Stephens (1927), maintains that in case of haemo-lymph glands it would appear as if all the red cells were fated to be destroyed in the reticulum which traverses the blood sinuses or lacunae . . . all transitions from red cells to debris and pigment can be found.

These few references about the structure of the haemo-lymph gland more or less support the opinion expressed by the writer that these nodules in the liver resemble haemo-lymph glands.

How far do these nodules in the liver seen in splenectomized ruminants resemble those seen by others in the peritoneal cavity of man, etc., after splenectomy? M. B. Schmidt (1914) made interesting observations in the liver of splenectomized white mice. A short while after splenectomy cell multiplication takes place in the liver, which soon (probably four to five weeks) forms small nodules which morphologically resemble spleen pulp tissue. The cell accumulations are formed along blood-vessels, and seem to be derived in part from the capillaries, i.e. the stern cells. Schmidt furthermore showed that this new tissue assists in the destruction of erythrocytes in place of the spleen, if not quantitatively, at least qualitatively, but in this tissue the process is slower than in splenic tissue.

Seifert further tested the function of this newly formed tissue, especially as regards its capability of hoarding pigment. He showed that hoarding took place in the intrahepatic multiplication of stern cells of guinea-pigs previously splenectomized. In two human beings affected with pernicious anaemia he noticed some time after splenectomy (in one case after eight weeks, in the other six and a quarter years) hypertrophy of the stern cells, but no increase of these cells. He found *no* compensatory new formation of splenic tissue, which was described by other authors in the omentum.

Schmidt also refers to similar changes in the human liver to those seen in mice after splenic insufficiency (e.g. extensive contraction of the spleen as result of infarction), with the formation of accessory spleens in the large and small omentum and cell foci in the liver.

Bittner (1913) observed in rabbits after splenectomy extensive cell accumulation, partly with the formation of germ centres, in the kidneys, liver and lungs. He also says that he did not observe in his animals the compensatory formations and splenoid tumours in the peritoneum as described by Beneke and Faltin. Unfortunately the writer could not obtain the literature in which these observations are referred to.

Catsaras (1928) quotes the observations of Beneke, viz., of a case in man four years after splenectomy where numerous firm greyish-black nodules appeared in the peritoneum. They resembled splenic pulpa. Beneke was of opinion that this was due to the growth of traumatically shattered splenic " rests " as a result of the operation of splenectomy. According to Catsaras, Faltin observed in a boy six years after splenectomy numerous spleen-like nodules, about the size a cherry, in the peritoneum during appendectomy. Micro- these nodules consisted of lymphoid tissue made up of a f follicles. Catsaras regards these as derived from resting ntres in the peritoneum, which become stimulated and grow withdrawal of splenic function.

uld therefore appear that haemo-lymph-like or spleen-like ave sometimes been observed to occur in the abdominal er splenectomy. In all the cases recorded above, except Schmidt, none of these nodules made their appearance ver substance. According to Schmidt these nodules in of white mice morphologically resembled splenic pulpa. r, however, is of opinion that the nodules in the liver, those encountered in the goat and sheep, closely resemble ure of a haemo-lymph gland. It is intended, however, to further pigment hoarding studies, etc., in connection with ectomized sheep and goat still alive, in order to obtain a iled study of the finer structure of these nodules seen in the ruminants.

CONCLUSIONS.

full description is given of the multiple nodules observed ers of a number of ruminants that died or were killed about s after splenectomy.

hese nodules were not of the nature of a neoplasm, but of a ed tissue, closely resembling the structure of a haemo-lymph

he literature is discussed in respect of more or less similar n man and other animals after splenectomy.

ADDENDUM.

e haemo-lymphoid-like nodules of the liver of sheep 8428 n 8231) here and there an irregular network of strands could ied. In places they had the character of short rods lying and sometimes these rods appeared segmented. With the e method these strands assumed a diffuse blue colour. One emo-lymph glands of this sheep showed similar strands in of multiple networks. These were chiefly situated along hery of the glands. The presence of foreign body giant ociated with smaller pieces of these rods, were also noted. instances these giant cells completely encircled these rods.

ral observers have noticed similar structures in the spleen and considered the thread-like structures to be mycelium. ther hand, many German pathologists, as well as recent ors, believed that the filamentous structures were degene- ne fibres. C. H. Hu, H. A. Reimann, and T. G. Kurotchkin *roceedings of the Society for Exp. Biol. and Med.*, Volume o. 6) came to the conclusion that, although the relationship fibres, and their staining reactions and morphology are different from the fungi cultivated from the same spleens. Unfortunately, the writer did not have fresh material to ascertain whether the strands seen by him were of the nature of a fungus. The impression obtained from the sections of the haemo-lymph gland and haemo-lymphoid-like nodules in the liver of sheep 8428, was that these strands were most probably degenerated tissue fibres which had become impregnated with iron pigment. This is probably the first instance in which such strands have been recorded to occur in a haemo-lymph gland and in these haemo-lymphoid-like nodules in the liver of a splenectomized sheep.

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APPENDIX.

Under this will be included:

(A) Post-mortem reports of the cases referred to in the text, together with microscopic descriptions, etc.

(B) Photographs, etc.

BOVINE 893: SPECIMEN 7408.

History.—15.12.24, splenectomized. 18.12.24, relapses of piroplasmosis, and subsequently anaplasmosis, from which the animal recovers completely. 10.11.27, dies in trypanosome experiment.

Post-mortem.—Black cow, about three and a half years old. Died during the night. General anaemia; emphysema and hyperaemia lungs. Pigmentation, slight degeneration and multiple circumscribed haemo-lymph-like nodules in the liver. These nodules in the liver vary in size from $\frac{1}{4}$ to 1 inch in diameter; they are completely circumscribed, slightly raised above the cut surface of the liver, dark reddish fleshy appearance, almost the colour and consistence of a red thrombus. These nodules can more or less be completely enucleated, leaving a fairly smooth greyish-white cavity, resembling the lumen of a vein, except that it does not appear to be in direct communication with any large vessel; ascites; hydrothorax; hyperplasia of lymphoid tissue; hypertrophy of haemolymph glands; absence of spleen.

Cause of Death.—Sequel trypanosomiasis.

Microscopical Appearances.

Liver.—Here and there irregular round circumscribed foci varying in size from a pea to a walnut. These foci are made up of erythrocytes, which lie in a system of blood-vessels associated with round cells, reticulo-endothelial cells, and a good deal of pigment. Some of the pigment is haemosiderin, whereas a sprinkling of fine granules does not react with berlin blue stain. The whole focus is supported by a delicate stroma of connective tissue and is surrounded by a connective capsule, which, however, in places shows a break in the continuity, so that the focus mass becomes confluent with liver substance without any line of demarcation. This capsule on the inside seems to be lined with an endothelial layer, and it may be noted that with Weigert stain the elastic tissue in the capsule assumes a regular arrangement. It may be pointed out that, in the interior of these foci, small blood-vessels can be identified. With the berlin blue method it will be seen that the distribution of the pigment is in cellular elements resembling those associated with haemosiderosis in the spleen. The haemosiderosis in the nodule is prominent and extensive, whereas in the liver substance a few scattered desquamated stern cells with haemosiderosis are met with. The rest of the liver substance shows an extensive vacuolated appearance which with sudan III proves to be droplets of fat. These fatty changes affect the lobules uniformly.

Kidney.—Slight fatty changes in the epithelium of the tubules. Small amount of haemosiderin present.

Lung.—Slight haemosiderosis.

Lymph Glands.—No specific changes seen.

Haemo-lymph Glands.—Hypertrophied, but no specific changes seen.

BOVINE 1034: SPECIMEN 8186.

History.—20.1.25, splenectomized. 10.2.25, relapse of piroplasmosis and subsequently anaplosmosis, from which the animal recovers completely. 5.6.28, dies of piroplasmosis when tested with infected blood as regards its immunity.

Post-mortem.—Black ox, about three years; interim about $\frac{1}{4}$ hour; general anaemia; icterus; haemoglobinaemia; haemoglobinuria; extensive interstitial emphysema in both lungs and peritracheal tissues; extensive pigmentation in kidneys; slight extravasations on both endocards; enlargement and hyperaemia of lymphatic glands; lipomatosis; acute catarrhal gastro-enteritis, with multiple haemorrhages; absence of spleen; degenerative changes liver, and multiple haemo-lymphoid-like nodules in the liver. These nodules become apparent through the capsule of the liver as dark red, almost purple, circumscribed areas. On section these nodules appear to be made up of a dark red, almost black, homogeneous mass and appear to be completely encapsulated; consistence almost like that of a firm thrombus material. In the substance of the liver there are a number of smaller nodules, about the size of a pea, also completely circumscribed.

Cause of Death.—Piroplasmosis.

Microscopical Appearances.

Liver (first piece).—Two large circumscribed encapsulated nodules, made up of an irregular system of blood sinuses, lined in places by endothelial cells. In the larger nodule here and there indefinite streaks of lymphoid tissue can be identified; associated with the system of blood sinuses there appears to be an arrangement of cells (like reticulo-endothelial cells) staining dark blue in colour with berlin blue (haemosiderosis). Along the periphery of the liver lobules there is a bile stasis and a good deal of haemosiderosis, associated with the stern cells. A hyperaemia was also noted in the liver lobules.

Liver (second piece).—Vacuolation of liver cells more prominent, and the liver tissue affected in patches. Here and there in the liver substance are pools of blood causing atrophy of liver tissue. The capillaries are distended with blood. In places the pools of blood are frequent and extend over a large number of lobules.

Sections of the Liver Nodules.—Large amount of blood in the spaces; only a few (three) small circumscribed lymphoid masses present (follicles?), but here and there irregular strands of lymphoid tissue can be identified. With haemalum-eosin stain, scattered through the blood sinuses are cells resembling reticulo-endothelial cells, and the majority of them contain a minute yellowish-brown granular pigment.

With berlin blue the bulk of this pigment remains yellow-brown, whereas a small amount is haemosiderin. Here and there also rod-like homogeneous masses of haemosiderin (cf. Specimen 8231).

Kidneys.—Haemoglobinaemia, but more extensive haemosiderosis, associated with the epithelium of the tubules and the lumen, in the former as intensely dark-bluish stained granules, and in the latter as diffuse light bluish homogeneous masses. The convoluted tubules nearer the capsule of the kidney were more extensively affected.

Lungs.—Hyperaemia; fairly extensive haemosiderosis in the capillaries, in places in the form of minute granules.

Hypophyses, Intestine, Pancreas, Cerebellum, and *Bone Marrow.*—No specific changes seen.

Lymph Glands I and II.—Good deal of blood in the sinuses. Here and there haemosiderin in the form of clumps of a dark greenish-blue colour. They occur in clusters and in places give one the impression that the haemosiderin is in the form of cylindrical masses in the lymphoid follicle, whereas the sinus cells appear only slightly affected with haemosiderosis? Note that the blood in the capillaries shows about 100 per cent. infection with piroplasms.

Lymph Gland III.—Erythrocytes in sinuses, and a good deal of granular haemosiderosis in the sinus cells.

Lymph Gland IV.—Same as lymph gland I and II; large amount of blood in the sinuses. With Giemsa the pigment stands out as conglomerated masses of light greenish yellow brown homogeneous masses of different sizes.

Lymph Gland V.—Blood in sinuses. Pigment masses, i.e. haemosiderin as the above, in some follicles.

BOVINE 870: SPECIMEN 8016.

History.—30.4.25, splenectomized, followed by relapse of anaplasmosis, from which animal recovers. 3.5.28, dies of piroplasmosis when subjected to natural infection.

Post-mortem.—Red ox, aged, died in the camp where the post-mortem was conducted. Carcase showed signs of having been tampered with by dogs, etc.; decomposition changes; general icterus; cachexia; general anaemia; absence of spleen.

Liver.—Normal in size and shape; capsule is smooth and glistening. On the parietal and visceral surfaces presence of numerous circumscribed reddish purple nodules noted, about 2 mm. to 6 mm. in size, scattered over the surface, and in the substance of the liver. Rest of liver tissue, except for p.m. changes, shows nothing unusual.

Cause of Death.—Acute piroplasmosis.

Microscopical Appearances.

Liver.—Hyperaemia with evidence of slight atrophy of the columns of liver cells. One of the nodules referred to above is seen in the section as a well-circumscribed area, separated from the liver tissue by a well-formed connective tissue capsule. The nodule is made up of a vascular system filled with blood, and lined by endothelials supported by a delicate connective tissue stroma. Interspersed in this stroma are accumulations of lymphocytes which in places resemble a kind of follicle. In the stroma there is evidence of pigment metabolism.

In the nodule well-defined blood-vessels can be identified, in virtue of the regular arrangement of elastic fibres, either longitudinally or circularly. The nodule is surrounded by a well-defined capsule, which contains regularly arranged and distributed elastic fibres and fibrillar connective tissue, the thicker fibres are on the outside of the nodule.

With berlin blue there is a characteristic appearance of haemosiderin granules having a linear arrangement along the blood-vessels in the liver substance and in the nodule.

Kidney.—Signs of haemoglobinaemia, but marred by putrefactive changes.

Lymph Gland.—Good deal of blood in the sinuses, especially in the cortical portion.

Myocardium.—A few sarcosporidia present.

BOVINE 1032: SPECIMEN 7852.

History.—21.4.25, splenectomized; 26.4.25, relapse of piroplasmosis and subsequently anaplasmosis; 20.3.25, dies of piroplasmosis when exposed to natural infection.

Post-mortem.—Red and white ox, full mouth. General anaemia; general icterus; haemoglobinaemia; haemoglobinuria; enlargement of lymphatic glands, some with pigmentation, and hyperaemia; haemal lymph glands more prominent than usual; adiposity; multiple necrosis of adipose tissue, e.g. fat capsule of the kidney; absence of spleen; pigmentation and degeneration of kidneys; hydropericardium.

Liver.—Enlarged; edges thickened; reddish brown; multiple dark reddish purple nodular-like formations; although irregular in outline, they are well circumscribed. In places some of them are confluent; some nodules show up on the parietal surface, others on the visceral surface. The liver on section also shows nodules in the substance. They are of a homogeneous nature, and protrude above the cut surface of the liver substance. Rest of liver substance on section is light reddish-brown in colour, and shows well-marked bile stasis in the periphery of the lobule; consistence not reduced. Acute catarrhal enteritis; localized ossification in media of aorta.

Cause of Death.—Acute piroplasmosis.

Microscopical Appearances.

Liver.—Bile canaliculi in the periphery of the lobules distended with bile; the nodules show a system of thin-walled spaces containing a large amount of blood. In the delicate connective tissue stroma between these spaces are large phagocytes containing a granular brownish-yellow pigment. Good many lymphocytes present, but no well-defined follicles seen. In some sections the lymphocytes are more prominent and occur in masses. With berlin blue stain a large number of the phagocytes show the presence of haemosiderin, whereas a good number of them reveal a dark brown granular pigment.

The nodule occupies an area which is equivalent to several lobules. The capsule of the nodule is fairly thin and infiltrated with numerous round cells and erythrocytes which seem to run into the central mass without any definite endothelial or other lining to the capsule.

Lymph Gland.—In some of the sinuses red cells and neutrophiles.

Lung.—Capillary network filled with neutrophiles; alveoli show a very slight mixed pneumonia, but mainly catarrhal; there is fairly extensive haemosiderosis in the lung tissue.

Kidneys.—Tubules and glomeruli contain a homogeneous pink staining substance.

Pancreas.—In between the acini here and there large masses of adipose tissue. (*N.B.*—The changes in the pancreas and the fat necrosis observed in various parts of the abdominal cavity will be dealt with in a separate paper.)

BOVINE 894: SPECIMEN 8055.

History.—17.3.25, splenectomized, followed by relapse of anaplasmosis; 21.5.28, died of piroplasmosis when exposed to natural infection.

Post-mortem.—About four years; condition fair. Oedema of the lungs; degeneration myocardium; hoemoglobinaemia; haemoglobinuria; general pigmentation, due to trypan-blue injection; absence of spleen (splenectomized).

Liver.—Markedly swollen; on the surface several bright dark red mulberry-like nodules can be made out, well circumsribed and easily differentiated from the brown liver tissue. These nodules are about 1 to 2 cm. in diameter, and also occur in the substance of the liver tissue. The liver lobules show yellow-brown pigmentation in the periphery. The consistence of the liver is reduced and it is more friable.

Cause of Death.—Acute piroplasmosis.

Microscopical Appearances.

Liver.—Good deal of blood and cellular elements in the intralobular capillaries. They appear widened and the columns of liver cells atrophied. The periphery of the lobules also show a good deal of cellular infiltration. Some lobules show the presence of a good deal of fat, whereas in a limited number of lobules there is a slight necrosis affecting a limited number of liver cells around the central vein. Fairly well-marked haemosiderosis associated with the stern cells. In places Glisson's capsule shows the accumulation of round cells.

In the nodule there is a system of blood-vessels and associated with its walls are reticulo-endothelial cells showing well-marked haemosiderosis. There is also a good deal of yellow-brown granular pigment present. Some of the sections of the nodules seen in the liver show lymphoid tissue collected into well-circumscribed follicles. In some nodules three to four such follicles can be identified.

Kidneys.—Haemoglobinaemia and haemosiderosis.

Myocardium.—A number of sarcosporidia seen and fairly well-marked fatty changes present.

Lymph Gland.—Note the hoarding of trypan-blue granules associated with the reticulo-endothelial cells.

Lungs.—Hyperaemia and oedema, and fairly extensive haemosiderosis.

Bone Marrow in Rib.—Sections not very satisfactory, but no formation of lymphoid follicles detected.

SHEEP 10944: SPECIMEN 7765.

History.—11.5.25, splenectomized; 16.7.25, relapse of anaplasmosis; 21.2.28, died unexpectedly in tar application experiment.

Post-mortem.—General adiposity; multiple fat necrosis; extravasations and petechiae epicardium; degenerative changes in kidneys; absence of spleen (splenectomized).

Liver.—Enlarged, slight yellow-grey colour, a few dark reddish areas present in the parietal surface of the liver. On section these are found to extend into the substance of the liver and are about 2 mm. in diameter; rest of the liver substance is soft and friable; lobulation distinct. A very few of these dark-red nodules present in the liver substance.

Cause of Death.—Extensive fatty changes in the liver, probably sequel to application of tar to the skin.

Microscopical Appearances.

Liver.—Here and there associated with Glisson's capsule are a few well-circumscribed nodules, made up of a delicate connective tissue stroma and a large amount of blood, which lie in endothelial lined channels. Interspersed in this stroma are large cells which contain a brownish granular pigment in clusters. Here and there in this nodule well-developed lymphoid follicles can be made out. The pigment referred to above is haemosiderin and is present in large amounts. In the liver tissue itself, there is a very little haemosiderin present in a few lobules surrounding the nodule, whereas the rest of the liver tissue shows no pigment. The haemosiderin in the nodule is in the form of blobs. With the Giemsa stain these cells, with pigment, embedded in the stroma of the nodule, resemble stern cells, and in them the remains of erythrocytes can definitely be made out in all stages of disintegration, from recently phagocyted erythrocytes of the usual size to mere pigment granules. The rest of the liver tissue shows a certain amount of fatty infiltration.

Kidneys.—Fairly extensive fatty infiltration, especially in the tubules of the cortex.

Myocardium.—Shows a moderate amount of fatty infiltration.

Pancreas.—No changes seen.

Lymphatic Gland.—Slight anthracosis and hyperaemia.

Lungs.—Here and there in the alveolar walls are dark greyish-brown pigment masses (tar).

SHEEP 8428: SPECIMEN 8231.

History.—3.7.24, splenectomized; 11.7.24, relapse of anaplasmosis; 19.7.28, killed, no signs of ill-health. A few Gonderia in the blood.

Post-mortem.—19.7.28, merino hamel, aged, condition good; aspirated blood in both lungs; multiple haemo-lymphoid-like nodules in the liver and associated with the capsule, where they appear as reddish-purple circumscribed areas, varying in size from a pin's head to those about 1 cm. in diameter; they have a fleshy appearance and are raised above the cut surface of the liver substance. These nodules reveal in their substance lymphoid follicles as greyish-white transparent circumscribed masses. Their number varied according to the size of the nodule. In the smaller ones only one follicle could be identified; absence of the spleen; adiposity.

Cause of Death.—Killed in order to ascertain whether the above-described nodules were present in a clinically healthy animal.

Microscopical Appearances.

Liver.—With sudan III numerous small droplets are seen evenly dispersed over the whole section; in the intralobular vessels the reticulo-endothelial cells stand out more prominently as bluish streaks. The contour of the lobules is not regular, due to the accumulation of cellular elements, mainly of the lymphoid type, in Glisson's capsule. There is no proliferation of bile-duct epithelium. In places these cell accumulations in Glisson's capsule assume the character and dimensions of lymphoid follicles.

In the nodules seen, there is a system of blood-vessels which are associated with cells probably of the reticulo-endothelial system. These nodules are well defined, but not encapsulated. The system of blood-vessels is not demarcated from the liver substance. With the berlin blue stain, a large amount of haemosiderin is found to be present in the liver cells. This is especially prominent in those cells near the periphery, and also in liver cells around the nodule where they stand out prominently as a fairly broad zone.

Extensive haemosiderosis is also present in the nodule, and seems to be associated with cells probably of the reticulo-endothelial type. In places the periphery of the nodule shows the presence of a number of macrophages containing pigment. In the nodules numerous short narrow tubule-like rods form a sort of network. They stain intensively with berlin blue. Associated with these structures are giant cell formations of the foreign body type, which are arranged in the form of a circle, surrounded by round cells. With Giemsa these rods stain light green, and give the impression that they are made up of " degenerated " tissue fibres. ...

Very characteristic in these liver nodules are the formations of lymphoid follicles, each with a germinal centre. These follicles can in no way be distinguished from those observed in haemo-lymph glands. These follicles are in no way associated with a haemosiderosis.

Kidney.—Here and there the convoluted tubules show a granular haemosiderosis in the epithelium.

Lung.—In places the capillaries contain a good deal of blood. There is, associated with the capillary walls, a well-marked haemosiderosis.

Myocardium, Central Nervous, Tongue, Adrenal.—No specific changes seen.

Bone Marrow.—Note the characteristic formation of definite lymphoid follicles, like those seen in the haemo-lymph gland. The bone marrow can be distinguished from the latter, however, by the presence of megakaryocytes and precursors of leucocytes and erythrocytes.

Lymph Glands I and II.—The lymphoid follicles are prominent, especially so towards the medulla. The so-called " germinal centres " also stand out prominently. No haemosiderosis detected in the sinus cells.

Haemo-lymph Gland.—The distribution of follicles and sinuses containing a good deal of blood is very similar to the nodules seen in the liver. Even the isolated patches of short broken rod-like structures, staining intensively with berlin blue, and referred to in the nodules of the liver, were also detected in the haemo-lymph glands. Giant cells were also present. The distribution of haemosiderin was also more or less the same.

GOAT 8304: SPECIMEN 8230.

History.—30.5.24, splenectomized; 24.11.24, reaction of anaplasmosis; 19.7.28, killed. Showed no signs of ill-health.

Post-mortem.—19.7.28, condition very good; ewe; aged; adiposity; the liver revealed through the capsule several discrete circumscribed nodules, up to the size of a split-pea, dark red in colour; about half a dozen detected; they have a fleshy consistence, with a mulberry-like surface. These nodules also showed the presence of circumscribed transparent greyish-white lymphoid masses.

Cause of Death.—Killed in order to demonstrate the presence of the above-described nodules in the liver.

Microscopical Appearances.

Liver.—Well formed nodules, circumscribed, and each contains three to four well-defined and developed lymphoid follicles. Smaller lymphoid masses are arranged in the form of strands. The rest of the nodule is made up of a system of blood sinuses, but is not present to such large extent as the lymphoid tissue. Associated with the walls of the sinuses are masses of a dark yellow-brown granular pigment in clumps. In this case no other accumulations of cells seen in connection with Glisson's capsule. With the berlin blue stain not much haemosiderin present in the liver substance, whereas the nodules reveal haemosiderin in the form of clumps. These clumps vary from a conglomeration of a few granules to large masses. With the Giemsa stain, well-marked and well-defined erythrophagocytosis can be made out in what look like reticulo-endothelial cells.

Haemo-lymph Gland.—A few well-defined lymphoid follicles occur around the periphery of the gland. A good deal of blood in the sinuses of the medulla of the gland.

Kidney.—Slight hyperaemia, especially in the medulla. No haemosiderin detected.

Myocardium, Thyroids, Central Nervous System, Hypophysis, Adrenal, Tongue, and Intestines.—No specific changes seen.

Lungs.—Hyperaemia and haemosiderosis.

Lymph Gland I.—Well-marked brown granular pigmentation associated with the sinus cells. A large proportion of this pigment is haemosiderin.

Lymph Gland II.—Same as I, except that the hoemosiderin appears in clumps.

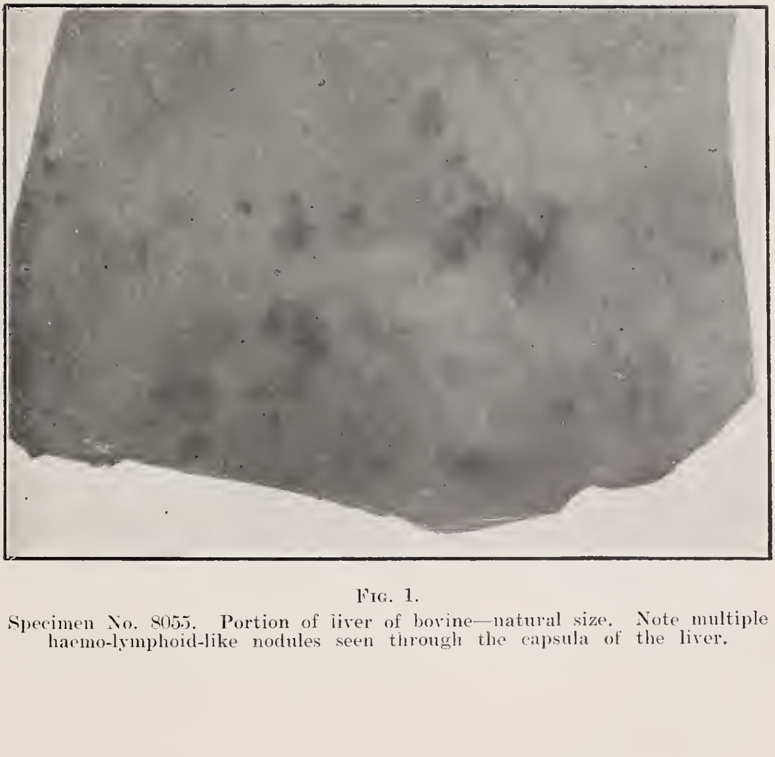

Fig. 1.

Specimen No. 8055. Portion of liver of bovine—natural size. Note multiple haemo-lymphoid-like nodules seen through the capsula of the liver.

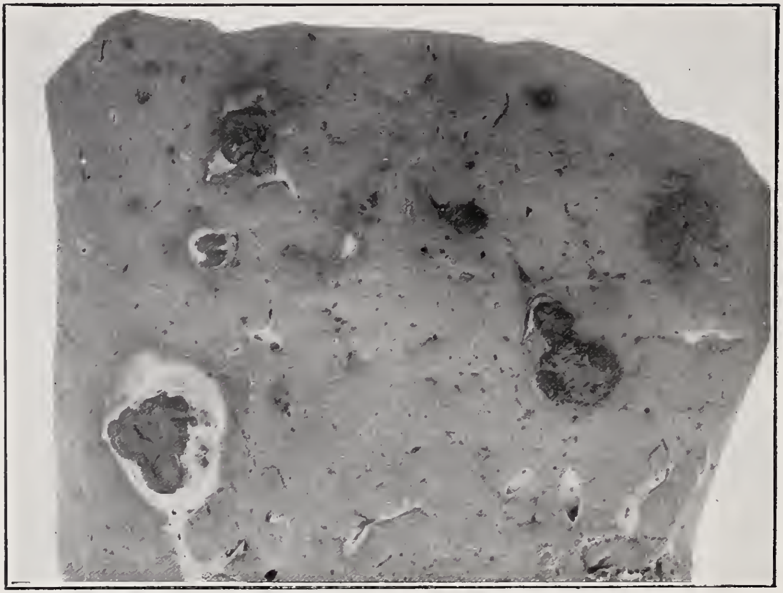

Fig. 2.

Specimen No. 8055. Portion of liver of bovine—natural size. Note the multiple haemo-lymphoid-like nodules in the substance of the liver.

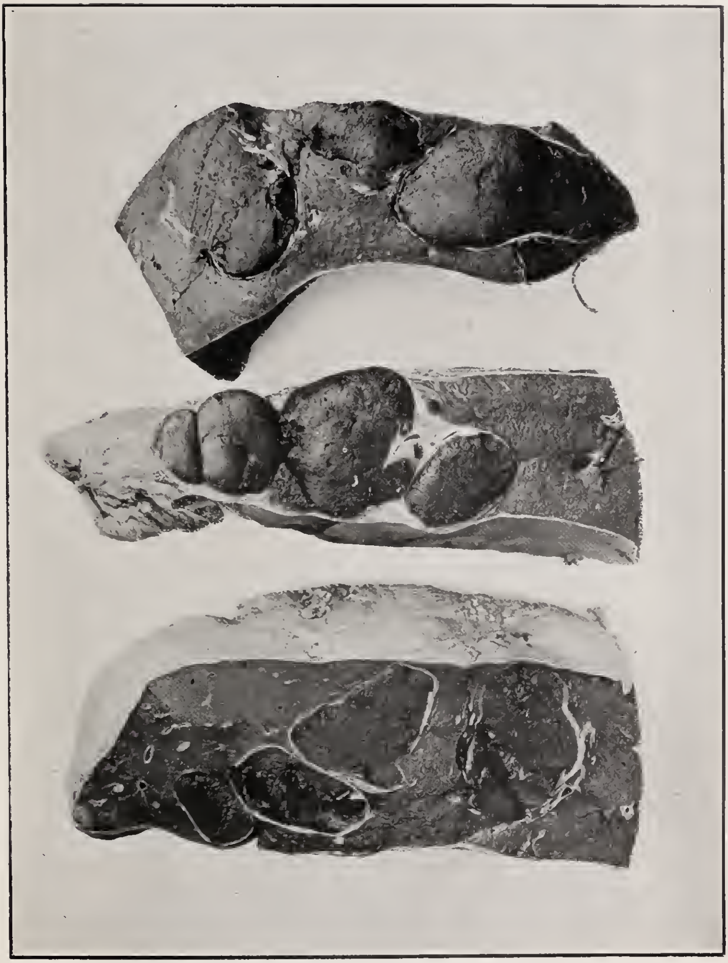

Fig. 3.

Specimen No. 8186. Portion of liver of bovine—natural size. Note the multiple haemo-lymphoid-like nodules in the liver substance. Some of these are distinctly raised above the level of the liver tissue.

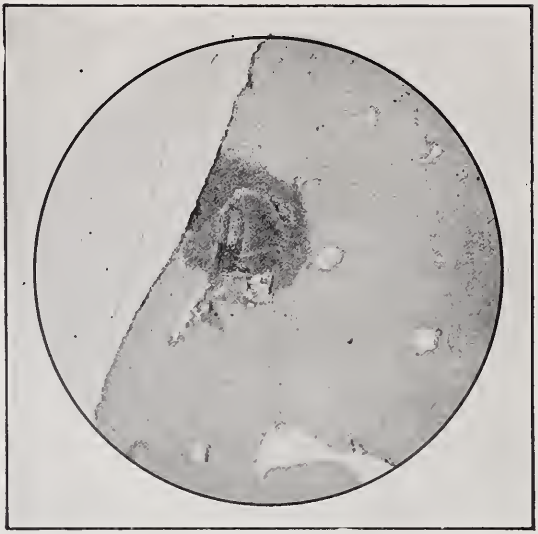

Fig. 4.

Specimen No. 7765. Liver of sheep (magnified 15 ×). Shows the presence of newly-formed haemo-lymphoid-like nodule, not demarcated from the liver substance by a capsule.

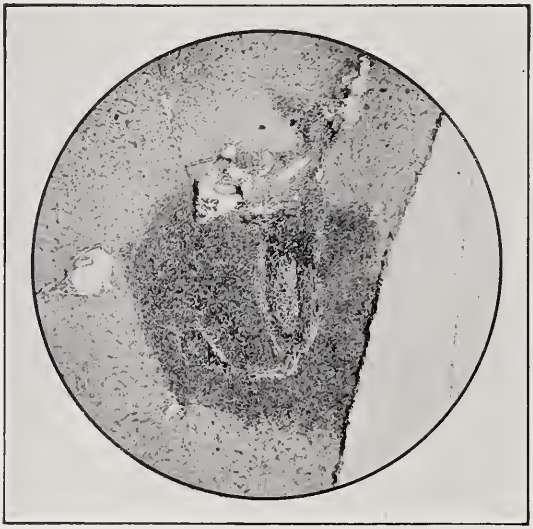

Fig. 5.

Specimen No. 7765. Liver of sheep (magnified 30 ×). Shows the presence of newly-formed haemo-lymphoid-like nodule, not demarcated from the liver substance by a capsule.

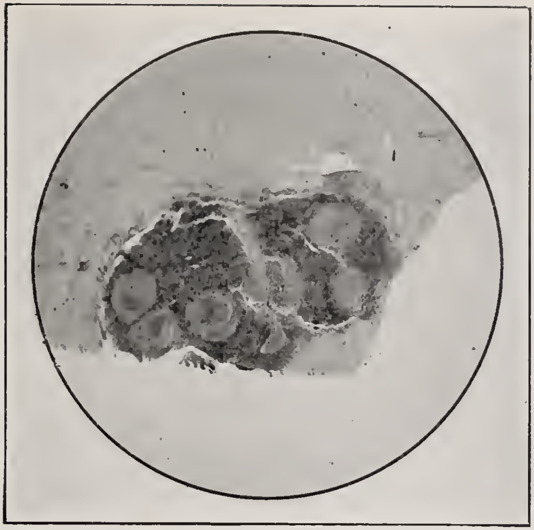

FIG. 6.
Specimen No. 8230. Liver of sheep (magnified 8 ×). Well-marked circumscribed haemo-lymphoid-like nodule in the liver substance, showing well-marked lymphoid follicles and strands of lymphoid tissue. Also note that the nodule is not well encapsulated in places, but projects into the liver substance.

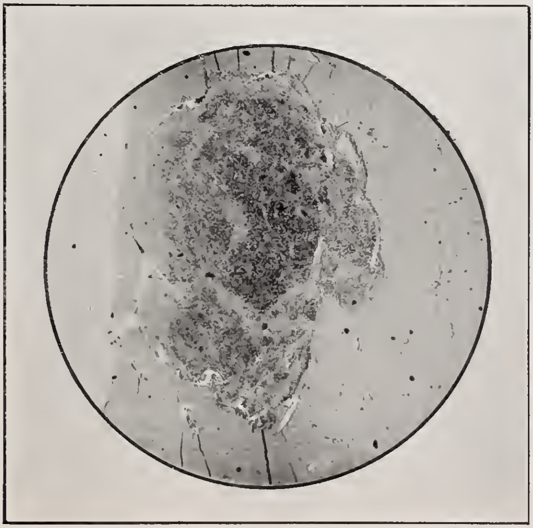

FIG. 7.
Specimen No. 7852. Liver of bovine (magnified 8 ×). Shows a newly-formed haemo-lymphoid-like nodule in the liver. Note the greyish-white lymphoid strands in this nodule.

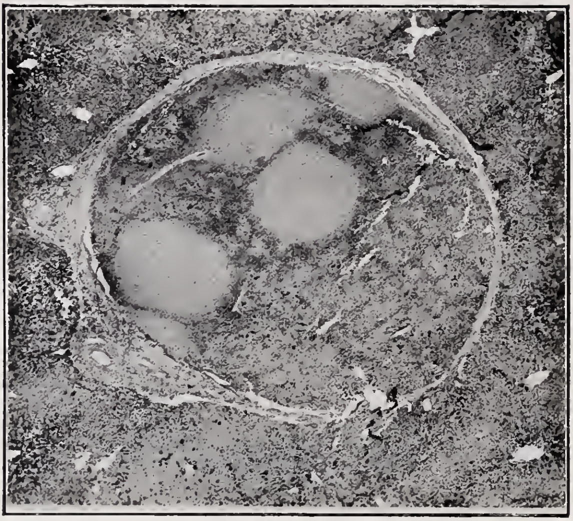

Fig. 8.

Specimen No. 8230. Liver of sheep (magnified 19 ×). Well-marked circumscribed haemo-lymphoid-like nodule in the liver substance, showing well-marked lymphoid follicles and strands of lymphoid tissue.

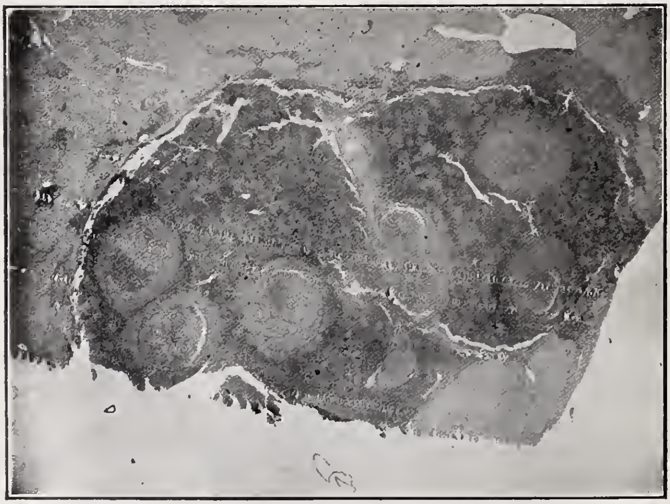

Fig. 9.

Specimen No. 8230. Liver of sheep (magnified 15 ×). Shows the presence of a well-formed lymphoid follicle in a part of a haemo-lymphoid nodule of the liver substance. Part of a liver is also shown. It will be seen that the nodule blends with the liver tissue without any definite line of demarcation.

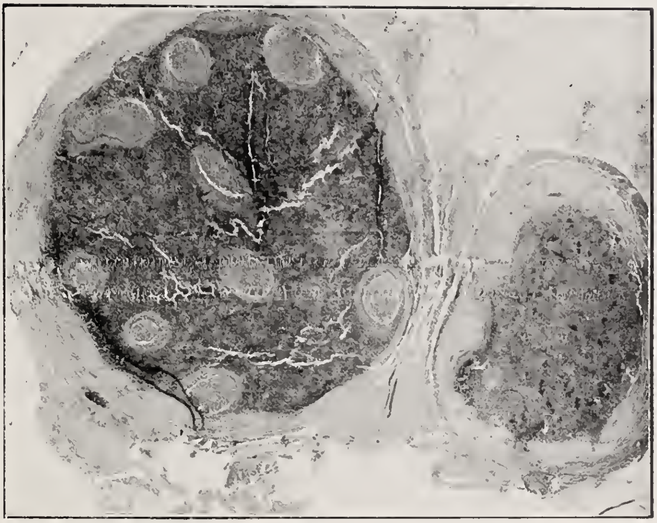

FIG. 10.

Specimen No. 8231. Haemo-lymph gland of a goat (magnified 15 ×). Note the distribution and character of the lymphoid tissue; also the rest of the substance of this gland. It will be seen that it resembles those haemo-lymphoid-like nodules seen in the livers of ruminants, especially in the case of the sheep and goat.

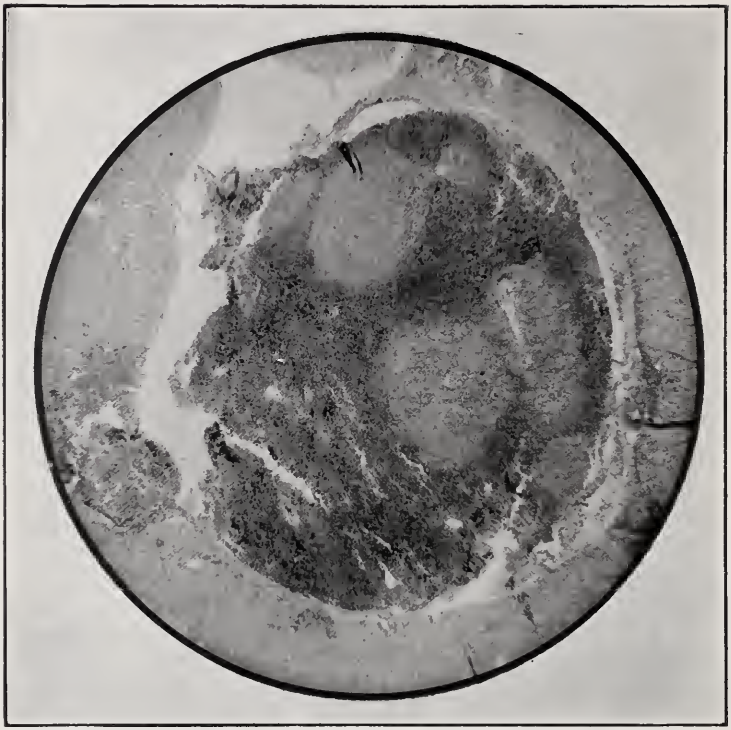

Fig. 11.

Specimen No. 8230. Liver of Sheep. (Magnified 38 ×.) Note the presence of the haemo-lymphoid-like nodule in the substance of the liver. This shows the presence of three well-formed lymphoid follicles. In the rest of the nodule the presence of haemosiderin granules can be noted as black-stained structures in the tissue. This is a photograph of a section stained according to the Berliner Blue method.

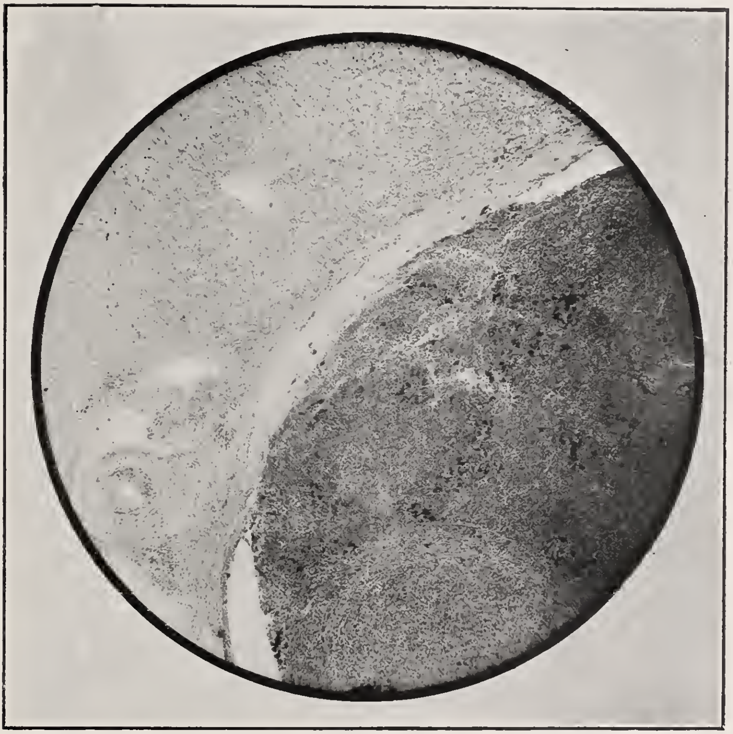

Fig. 12.

Specimen No. 8230. Liver of sheep (magnified 83 ×). Showing presence of a haemo-lymphoid-like nodule. These are haemosiderin granules, and it will be noted that they are only present in the nodule and not in the liver substance, which is also shown in part of the section. The nodule is demarcated from the liver substance by a well-defined capsule.

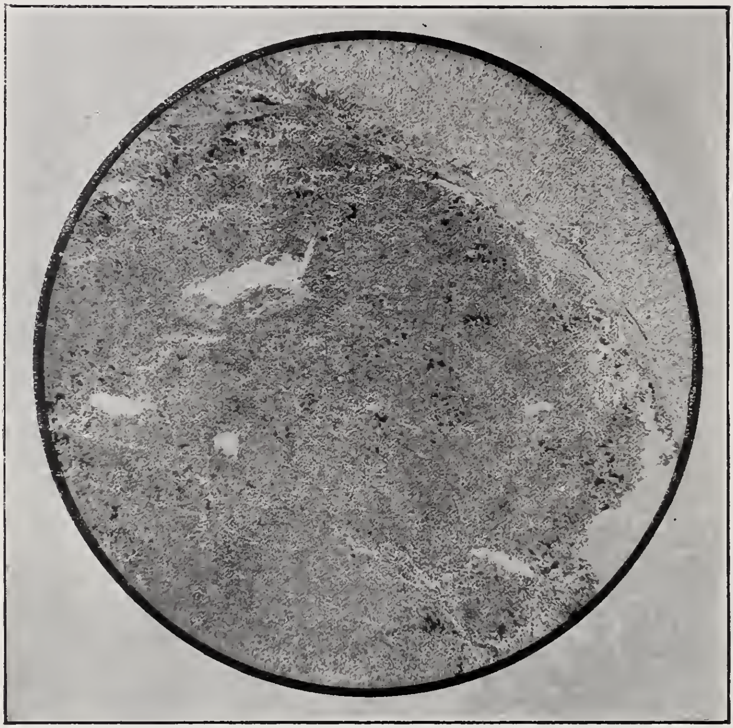

Fig. 13.

Specimen No. 8230. Liver of sheep (magnified 83 ×). Shows part of a haemo-lymphoid-like nodule, demarcated from liver tissue by a fibrous capsule. The haemo-lymphoid-like nodule shows the presence of half a follicle, and in the rest of the substance of the nodule are black-stained granules, due to the presence of haemosiderin. The section was stained according to the Berliner Blue method.

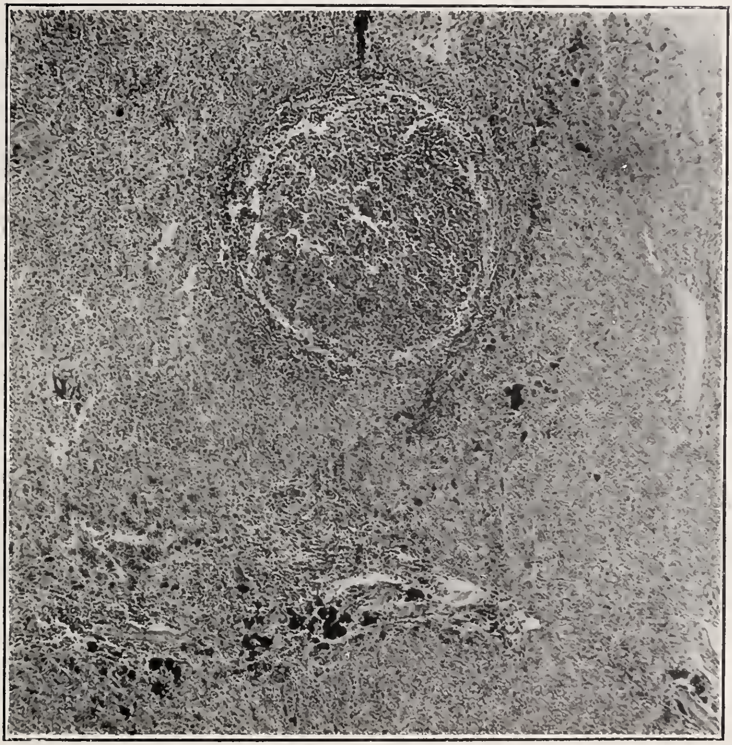

FIG. 14.

Specimen No. 8231. Liver of goat (magnified 135 ×). Note part of a haemolymphoid nodule together with parts of two liver lobules. The nodule is not demarcated from the liver substance by a fibrous capsule. In the nodule there is a well-formed lymphoid follicle. Note the peculiar haemosiderin pigmentation, referred to in the text.

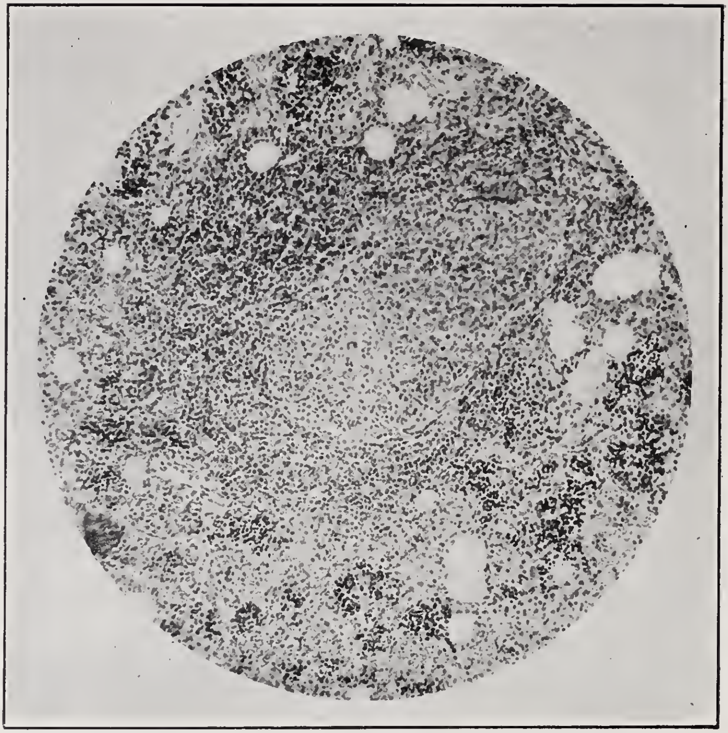

FIG. 15.

Specimen No. 8231. Section of the bone-marrow of a rib of splenectomized sheep 8428. Magnified 200 ×. Note the presence of a well-formed lymphoid nodule, of which there were many present in this section.

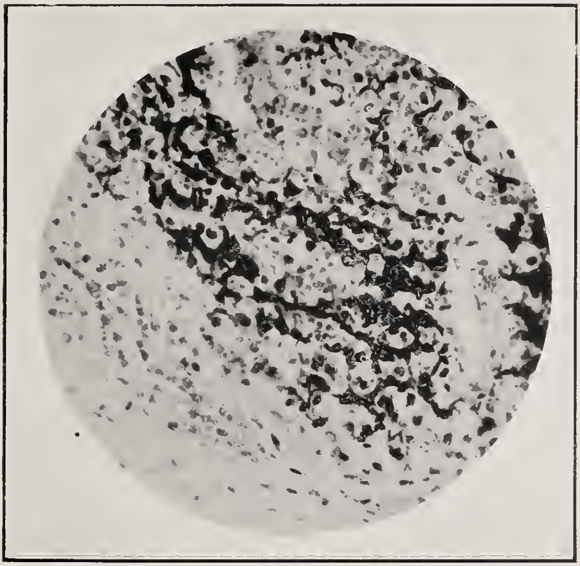

FIG. 16.

Specimen No. 8231. Section of an haemolymph gland of splenectomized sheep 8428. Magnified 350 ×. Stained Berlin Blue. Note the presence of the stained strands lying crossed. (See addendum.)

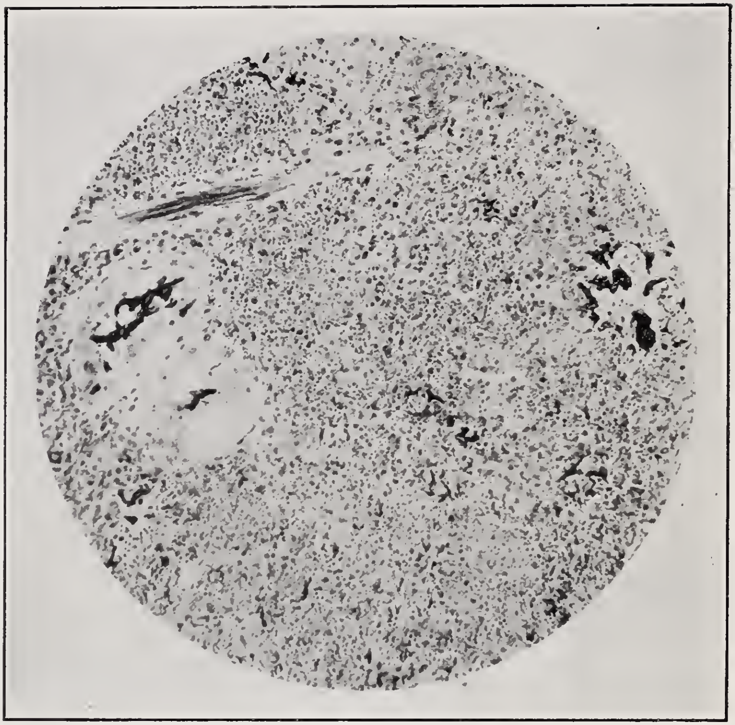

Fig. 17.

Specimen No. 8231. Section of an haemolymphoid-like nodule in the liver of splenectomized sheep 8428. Magnified 250 ×. Berliner Blue stain. Note the presence of the stained rods and those encircled by foreign body giant cells. (See addendum.)

15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

Are the Lesions of Jaagsiekte in Sheep of the Nature of a Neoplasm?

By G. DE KOCK, M.R.C.V.S., Dr.Med.Vet., D.Sc., Sub-Director of Veterinary Services, Onderstepoort.

THE pathology of Jaagsiekte in sheep has been studied by Mitchell (1915) and Cowdry (1925). According to Mitchell, jaagsiekte is essentially a chronic catarrhal pneumonia " . . . characterized by the presence of lymphoid follicles, which show a marked tendency to infiltrate the surrounding tissues . . . these may develop in connection with the bronchi or in the interalveolar tissue . . . there is also a bronchopneumonia, interstitial fibroid changes, bronchitis, and peribronchitis . . . " Mitchell found considerable variability in the occurrence of proliferations of the bronchi, and in a total of fifteen cases he described the proliferations as very marked in two, well-marked in three, present in two, not marked in two, not apparent in one, absent in two, while in the remaining three he made no comment regarding their presence or absence.

Cowdry is of opinion that the lungs of South African sheep, in a district in which jaagsiekte is appearing, differ from those of normal American sheep; " . . . the most significant difference, however, centres in the interalveolar tissue, which, in about 33 per cent. of the animals, is definitely thickened beyond the range of variation in this direction observed in American sheep . . . the thickenings begin with engorgement of the alveolar capillaries and accumulation of macrophages and of lymphocytes . . . many of the macrophages pass into the alveolar lumina and assume the appearance of typical epithelioid cells . . . these infiltrative and exudative changes are *primary* to the epithelial proliferations, which always arise in tissues modified in this way . . . "

It is important to note that the material collected by Cowdry for the study of lung tissue came from the following groups of sheep:—

(*a*) Known jaagsiekte cases;
(*b*) 38 sheep which died from bleeding in the preparation ot blue-tongue vaccine. These sheep came from a flock in which jaagsiekte was occurring from time to time;
(*c*) as controls, six sheep which died from heartwater.

In order to verify Cowdry's statements *re* the changes observed in the interalveolar tissue of the lungs of South African sheep, material from clinically healthy sheep from various centres was collected and studied. Mitchell's reference to the part played by the lymphoid tissue was also considered, and the possibility that Mitchell may have included under jaagsiekte cases of some other specific lung disease of sheep.

In order to obtain more information on various points raised by Mitchell and Cowdry in their studies of jaagsiekte, the writer set out to investigate the following points:—

(1) Do the lungs of sheep from infected areas show the changes which Cowdry maintains constitute the primary lesions?

(2) Can the "proliferations" of the respiratory epithelium be regarded as primary and of the nature of a neoplasm?

(3) What is the significance of the lymphoid "hyperplasia" in the lung, referred to by Mitchell, and was he in all instances dealing with one and the same disease?

(1) THE PRIMARY LESIONS OF JAAGSIEKTE.

Cowdry states that of the 38 sheep which were utilized for the preparation of blue-tongue vaccine, and which before death did not exhibit noticeable symptoms of jaagsiekte, four at autopsy showed distinct lesions characterized by variable degrees of epithelial proliferation. Very distinct differences were noted by him between the pulmonary tissue from the sheep in the middle west of America, and that from the South African sheep, themselves apparently free from the disease, but coming from flocks in which the disease was certainly enzootic. The changes in the lungs of the South African sheep centre in the interalveolar tissue referred to above.

It should be pointed out that it is not correct to conclude that the 38 South African sheep, amongst which Cowdry found lesions in 13, came from the same flock. During the period that these specimens were collected by Cowdry at Onderstepoort, about 2,000 sheep passed through the Institution for the preparation of blue-tongue vaccine, and these sheep did not all come from the same farm. They undoubtedly came from different flocks, in some of which jaagsiekte was probably present, while in others the disease was not occurring at all. Moreover, it should be remembered that these sheep from which Cowdry collected specimens were not clinically healthy, but showed reactions of blue-togne, and were accidentally bled to death for vaccine purposes.

A large number of lungs were collected by the writer from clinically healthy sheep. Some of these came from known jaagsiekte areas (e.g. Tweespruit), while others were from centres where jaagsiekte had not yet been diagnosed.

Lung specimens from clinically healthy sheep from the following centres were studied:—

Cape Province.—Tarkastad (7123); Colesberg (7126, 7131, 7132); Steynsburg (7127); Cradock (7128); Kimberley (7129).

Orange Free State.—Lindley (7122); Winburg (7125); Tweespruit (7398, 7409, 7424, 7488, 7516, 8091, 8109, 8183, 8319, 8382, 8454, 8561).

Transvaal.—Potchefstroom (7121); Standerton (7130); Volksrust (7516).

A large number of lung specimens from Onderstepoort sheep returned from Tweespruit and killed at the Pretoria Abattoir were also examined.

The lungs in all animals, killed by cutting the throat, showed the blood-vessels in the alveolar walls to contain a large amount of blood. Aspirated blood was usually present in some of the alveoli. It was found that this filling of the capilaries with blood, sometimes to a marked extent, also occurred in clinically healthy horses, dogs, bovines, etc., killed for the purpose of collecting specimens for histology. It will be very difficult in some of these sections to say whether the hyperaemia was actually due to disease or not. From these remarks it will be realized that the local congestion of the alveolar capillaries as a first stage in the formation of primary lesions could not be stressed to the extent as accepted by Cowdry.

The interalveolar infiltrations with macrophages and lymphocytes, and the penetration of the former in large numbers into the alveolar lumina, could not be substantiated in the lung sections of clinically healthy sheep from known infected areas. Nothing comparable with the photographs occurring in Cowdry's paper was observed in the writer's sections.

It would appear, therefore, that the papilliform proliferations, so characteristic of jaagsiekte, are not associated with the "primary lesions" as described by Cowdry. The writer managed to obtain cases of jaagsiekte in such early stages where proliferation had occurred without the presence of the infiltration of the interalveolar tissue with macrophages and lymphocytes.

More detailed information and discussion of these early proliferations seen in jaagsiekte will be given. The conclusion arrived at by the writer was that these proliferations were undoubtedly of the nature of a neoplasm. This was most characteristically borne out in three sheep with definite jaagsiekte proliferations, i.e. adenomata, which caused no sign of ill-health, and in which there was no complication of pneumonia.

The significance of the presence of foreign matter seen in many lung sections of sheep, some from probable non-jaagsiekte centres, is being further investigated. The presence of cells with sudan III stained granules, associated with the alveolar walls, should be mentioned here. It was present in almost every lung examined. In some instances as many as 20 such cells could be counted in the walls of a single alveolus. The granules were more or less of a regular size and appearance, and their colour varied from an intense brick-red to a dark brown. According to Fried (1928), physiologic investigations suggest that the lungs play a rôle in the metabolism of lipoids. This question is also being studied.

(2) THE SIGNIFICANCE OF THE EPITHELIAL PROLIFERATIONS IN JAAGSIEKTE.

According to Cowdry, the first indications of irregular growth consist of the enlargement and multiplication of cells bordering a portion of the lumen of an alveolus . . . the cells may assume a papillomatous arrangement. That they are actually of alveolar origin, and are not merely enlargements of the usual cubical cells situated at the point of termination of a bronchiole, was proved . . . the fact was revealed that they are in continuity on all sides with flattened alveolar epithelium.

In the material examined by the writer the first indication of an alteration in the very earliest lesions is the linear increase of cubical cells, i.e. multiplication of cubical cells on the wall of the alveolus. This leads to a bulging of the alveolus into the lumen, and papilliform formation. These multiplying cubical epithelial cells can easily be picked out in a section, and from then onwards all gradations can be identified to well-formed papillomatous formations.

The question arises whether the cubical cells formed are of the nature of a metaplasia of flattened alveolar epithelium, or a multiplication of pre-existing cubical epithelium. With reference to this, various points of importance about the histology of the lung may be briefly referred to. According to Szymononicz (1921), the lining of the respiratory tissue shows the following changes at various levels: bronchioles: cylindrical epithelium with cilia; bronchioles respiratorius: epithelium reduced in height and no cilia present; the alveolar passage, the infundibula (atria), and the alveoli possess a basal membrane, a capillary network, connective tissue and elastic fibres. . . .

According to Schäfer (1916), the alveoli are lined by large irregular flattened cells which form an extremely delicate layer, separating the blood capillaries from the air within the alveoli. Amongst the flattened cells are groups of smaller and thicker (cubical) epithelial cells. . . . Schaffer (1922) maintains that the respiratory epithelium which lines the alveoli shows in adults two types of cells: large thin plates without nuclei, and in between these, single or island-like groups of small polyhedral to cubical cells with nuclei.

Is it, therefore, necessary to assume a process of metaplasia? Is it not more probable that proliferations affect the cubical cells in the alveolar walls referred to above? These are the cells in possession of a nucleus and able to proliferate actively if stimulated. Sections have been studied in which these papilliform formations also affect bronchioles. This epithelium then assumes a columnar appearance without cilia (see figures). We therefore agree with Cowdry, viz., that the bronchiolar epithelium also contributes to these adenoma-like formations, which may sometimes assume large dimensions.

From the material studied, it would, however, appear that the majority of proliferations arise from alveolar epithelium. These papilliform formations grow and fill up the space made up of alveolar passage, atria, and alveoli. In this way multiple circumscribed nodules are set up. Each assumes a more or less regular circumscribed appearance, developing and growing into a lobule of the lung.

In the early cases, nodules, i.e. adenomata, in various stages of development could be identified. A number of these nodules may become confluent and in this way form larger nodules (see figures). The lung tissue may become so changed by these developing adenomata that it almost resembles, as Cowdry contends, mammary gland adenoma. The older jaagsiekte lesions may be associated with the presence of a good deal of stroma together with lymphocytes and histiocytes.

The plaque-like masses of growing cells attached to the inner wall of a bronchiole, described by Cowdry, have also been seen in the sections studied by the writer. In the mamma and ovary pure epithelial folds are formed without the presence of connective tissue in these sprouts. In the material studied by the writer, there was *no* evidence of metastasis in any of the other organs or tissues.

These adenomata may become associated with an acute catarrhal pneumonia (e.g. majority of the Tweespruit cases), to which the animal succumbs. A number of cases become complicated with an extensive chronic catarrhal pneumonia and fibrosis. This process usually invades the lung substance from the ventral to the dorsal aspect. Jaagsiekte foci make their appearance in the lung tissue as transparent, greyish white, circumscribed nodules, almost resembling lymphoid follicles. These nodules increase in number and size, and together with the pneumonia and indurations, which follow as complications, form firm, dense, very resistant greyish-white consolidated portions. In these the typical jaagsiekte foci can still be identified with the naked eye. Usually at post-mortem lungs are encountered in which two-thirds of the lung, i.e. in its ventral aspect, has been converted into a consolidated mass, whereas in the dorsal part isolated jaagsiekte nodules can still be identified in the lung tissue not much altered. These nodules are more frequent close to the consolidated portions. Above these consolidated portions the best material to demonstrate jaagsiekte foci should be selected. In a few instances necrosis and abscess formation were present in the consolidated parts. Sometimes these lung lesions become further complicated by a localized adhesive purulent pleuritis.

In the majority of cases studied by the writer the presence of the lymphoid cell accumulations, referred to by Mitchell, was not encountered. This will be more fully referred to below.

The proliferations which are so atypical in structure invade the apparently normal tissue so extensively and unrestrictedly without hope of regression that it would seem most appropriate to ascertain the view that they are true neoplasms, which are complicated by a terminal pneumonia. This would harmonize with the apparent absence of a direct causal organism in the lesion. If the tumour theory is correct, we would naturally look for some condition which would favour tumour formation. De Kock (1929) fully discusses the hereditary aspect of this question.

The writer had some difficulty in classifying these jaagsiekte neoplasms, which closely resemble the pictures given by Murphy and Sturm (1925). According to them, tumours in the lungs of mice occur as small white nodules, either single or multiple. The nodules are pearly white, varying in size from 1 to 4 mm. in diameter. As many as fifteen tumours were found in the lungs of one animal. According to these authors, the cells making up the tomours are fairly large, cubical or ovoid in shape, usually lying in a single layer on either side of a shred of stroma . . . there is some variation in the density of the tumour and in the amount of the stroma, but the general histologic type is uniform . . . as a rule the tumours are sharply demarcated from the surrounding lung tissue . . . the growths are identical in structure with those described as primary tumours of the lung in mice . . . they have been variously designated as adeno-carcinoma, papillary cyst-adenoma, adenoma . . . however, judging by the descriptions and published illustrations, there is but little variation amongst them, and they unquestionably represent a distinct type of epithelial tumour.

According to Borst (1922), the nearest approach to the lung tumours observed in jaagsiekte are those cystoma-ovarii papillare (described by him on page 330), especially as regards the microscopical picture, viz., complex labyrinth, best seen in a section through a cauliflower . . . the epithelial columns are seen in rich beautiful folds . . . the epithelium everywhere forms a simple layer of cubical or cylindrical cells.

From the above notes, and in view of the complete absence of metastasis in jaagsiekte, and the way in which the neoplasms are demarcated from the lung tissue, and in which epithelium and connective tissue harmonize, the writer is inclined to regard these neoplasms in jaagsiekte as of the nature of multiple papilliform cyst-adenomata.

(3) The Significance of the Lymphoid Hyperplasia and other Proliferative Changes in the Lungs of Sheep.

As pointed out above, Mitchell observed, in cases of jaagsiekte studied by him, nodules of lymphoid origin disseminated through the affected portions of the lung parenchyma, and occurring chiefly in connection with the bronchi, but also found isolated in the alveolar network. On the other hand, the epithelial proliferations did not occur regularly in the cases studied by him. He states that in some of the sections examined a condition was noted in which it would appear that proliferation of the bronchi had taken place . . . groups of small bronchial tubes could be seen cut across, each having a thin fibrous wall . . . this appearance may, however, be due to the section of a very much hypertrophied mucous membrane.

A careful examination of the lungs of a number of clinically healthy sheep was made, and it was characterized by the almost complete absence of lymphoid tissue in the lung. Only now and then a cross-section was obtained of a lymphoid follicle associated with a small bronchus or bronchiole.

In the great majority of lungs from Tweespruit sheep, affected with jaagsiekte, there was also a complete absence of lymphoid tissue. In a few cases a moderate number of lymphoid " infiltrations " was seen, besides the typical jaagsiekte lesions.

On the other hand, in a number of cases from the Experiment Station at Graaff-Reinet, lesions were identified which resembled those described by Mitchell, viz., (*a*) an extensive accumulation of round cells, chiefly of the lymphoid series; (*b*) an atypical proliferation of bronchiolar epithelium, i.e. not similar to those proliferations seen in the jaagsiekte sheep; (*c*) chronic catarrhal pneumonia, and the presence of giant cell formations in the exudates, present in the lumina of the alveoli (see figure). In one case some of the sections revealed a number of lymphoid nodules, some of these showing two and three germinal centres (see figure). The probability, therefore, is that in South Africa we may be dealing with two specific lung diseases of sheep.

(i) The one described by Mitchell, and of which the writer also obtained cases from the Graaff-Reinet Experiment Station, i.e. a lung disease characterized by lymphoid " infiltrations " or " hyperplasia "(?), apparent proliferative changes of the epithelium in places, and an indurative chronic catarrhal pneumonia. In the Graaff-Reinet cases in some exudates giant cell formations were present. Probably it is this disease that Mitchell was able to transmit so readily. That typical jaagsiekte adenomatous lesions may have been present in some of his in-contact experimental cases has been shown by De Kock to be most likely, due to the fact that some of the sheep may have had lesions of jaagsiekte, seeing that they came from a jaagsiekte-infected area (Middelburg, Cape).

(ii) Jaagsiekte in sheep characterized by the formation in the lung of multiple papilliform cyst-adenoma, complicated by a terminal acute or chronic catarrhal pneumonia.

It would appear that the progressive pneumonia in sheep of Montana very closely resembles the disease described by Mitchell. Marsh (1922) in his studies found that the principal change is the infiltration of the walls of the alveoli with small mononuclear cells and a similar peribronchial and perivascular infiltration . . . in places the infiltration is so extensive that the lumina of the alveoli are completely obliterated . . . in the consolidated portions of the lung there were found some places where the epithelial cells of a group of alveoli had changed to a cuboidal type, giving the appearance of cross-sections of a mass of bronchioles . . . giant cell formation is found in the cellular exudate in some cases.

In comparing Montana disease of sheep to jaagsiekte Cowdry maintains that the epithelial proliferation is usually less pronounced and resembles more a benign hyperplasia . . . the growth is distinctly less invasive and less neoplastic in appearance. Cowdry thinks that it would be difficult, through microscopic examination alone, to distinguish the lesions of McFadyean's (1920) cases of verminous pneumonia from jaagsiekte. It would, however, appear that the lesions described by McFadyean are again different to those cases studied by Mitchell, especially in respect of the following changes:— . . . (*a*) complete desquamation so that in some alveoli not a single epithelial cell remains in connection with the wall . . . (*b*) alterations of special interest are those which entirely transform the appearance of the alveolar epithelium, and thereby the aspect of the whole lung . . . i.e. alveolar epithelium has been renewed and the cells, instead of having the normal squamous or endothelial form, have assumed a plump cubical shape . . . in three or four air-cells there is an incomplete lining of cubical cells and in another portion of the lung the air-cells and infundibula have acquired a complete lining of similar cells . . . (*c*) in more advanced stages there is the transformation of the alveolar epithelium in certain parts, where it assumes a high columnar form, and this change has been accompanied by a multiplication which found a relief in the formation of ridges or villus-like growths projecting into the alveolar space . . . a productive process appears to have been going on in the mucous membrane leading to the development of ingrowths resembling those formed in the bile-ducts of a rabbit's liver in coccidiosis.

From the above notes, and the character of the lesions as shown in fig. 4 of McFadyean's article, it will be seen that probably a different process is involved in verminous pneumonia, namely, a very extensive desquamative catarrhal pneumonia. The productive changes and the formation of villus-like folds associated with the mucous membranes are of a different nature to those seen in the proliferative changes in the so-called adenomata of jaagsiekte, as described by the writer.

The question is now asked, whether one finds different types of " regeneration " or " proliferation " of respiratory epithelium in the lungs of sheep, viz.:—

(1) " Regeneration " of respiratory epithelium of a " productive " nature associated with different forms of chronic inflammations. This would be exemplified in the form of pneumonia described by Mitchell, the progressive pneumonia of sheep of Montana, and verminous pneumonia as studied by McFadyean. Kaufmann is of opinion that in chronic fibrous pneumonia the alveolar epithelium is often associated with a multiplication, partly covering the interalveolar connective tissue masses, the epithelium becomes thicker, cubical to cylindrical, and form, especially in old standing cases, glandular-like irregular proliferations, which may give a tumour-like impression (like the beginning of an adeno-carcinoma). The mechanism of growth here is maintained as the direct influence of an irritant, which is responded to by the tissues as long as the irritant continues. Here the new growth occurs in, and adapts itself solely to, the organ or tissues in which the irritant plays.

(2) " Proliferation " of respiratory epithelium, which is of autonomous character and seen in the growth of neoplasms.

It naturally becomes exceedingly difficult to draw a sharp line between tumour growth and certain other abnormal excessive localized hyperplasias and inflammatory tissue proliferations of similar appearances and behaviour, which stand on the border line of tumour growth.

This question of regeneration in connection with respiratory epithelium has been studied experimentally by Young (1928). Extensive and varied metaplasia of the serous endothelium of the visceral pleura and hyperplasia of the pulmonary epithelium can be produced by injecting into the pleural sac a mixture of sudan III and sodium cholate in olive oil. In further experiments on the production of hyperplasia in the alveolar epithelium of the lung of the rabbit, Young (1928) found that more active manifestations of increased cellular activity become apparent . . . the cells lining the marginal alveoli assumed a cubical form . . . these results tended to encourage the view that proliferative pneumonia were dictated by the direct action of the bile-salts upon the parent cells. . . .

Some of these experiments were repeated on sheep by the writer. In one case (specimen 8773) a large circumscribed vesicle formed in the lung substance as a result of the injection of the sudan III-sodium cholate emulsion directly into the lung. The margins of the vesicle showed productive changes and the formation of granulation tissue. There was an area on which there could be seen a peculiar type of proliferation of the epithelium which was not at all of the nature of those observed in jaagsiekte.

These experiments are proceeding and a further report will be published later on.

Conclusions.

(1) The local congestion of the alveolar capillaries as a stage in the formation of primary lesions of jaagsiekte could not be confirmed. Neither could the presence of interalveolar infiltrations, etc., as primary lesions, be substantiated in the lung sections of clinically healthy South African sheep from known jaagsiekte-infected areas, and those still believed to be free of the disease.

(2) Papilliform "proliferations" were identified in the very early lesions of jaagsiekte, without the presence of interalveolar infiltrations.

(3) The majority of the papilliform proliferations arise from alveolar epithelium. Bronchiolar epithelium, however, also contributed to the formation of papillamatous nodules.

(4) In view of the structure of these papilliform proliferations and their growth without hope of regression, these growths are most probably true neoplasms complicated by a terminal pneumonia.

(5) These neoplasms in jaagsiekte are probably of the nature of multiple papilliform cyst-adenomata.

(6) All gradations of developing adenomata have been studied. The lung tissue often becomes so changed by these proliferations that it resembles a mammary gland adenoma very closely.

(7) These multiple adenomata of jaagsiekte become complicated, either with an acute or chronic pneumonia, from which the animal sooner or later succumbs.

(8) The jaagsiekte nodules, together with the pneumonia and fibrosis, invade the lung substance from the cranio-ventral to the caudo-dorsal aspect.

(9) The jaagsiekte foci of the nature of pearly white circumscribed nodules, together with the pneumonia and fibrosis, form firm, dense, very resistant, greyish-white consolidations, which may involve the greater part of the lung.

(10) Above the consolidated parts of the lung isolated, well circumscribed jaagsiekte adenomata can always be identified.

(11) It is possible that in South Africa there may be two specific lung diseases of sheep, viz.:—

(*a*) a chronic indurative catarrhal pneumonia with lymphoid hyperplasias (?), or infiltrations (?), and atypical epithelial proliferations; and

(*b*) multiple papilliform cyst-adenoma complicated by a terminal pneumonia.

(12) The question of "proliferation" of respiratory epithelium, either associated with a chronic inflammation or a neoplasm, is discussed.

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APPENDIX.

(*a*) *Jaagsiekte Cases.*

Specimen No.	No. of Sheep or No. of Lab. Report.	Date of Arrival.	From Where.	Date of Death and Cause.	Remarks.	Pathological Histology.
3432	L.R. 7333	13/12/23	Belmont...........	—	Specimen..........	Very good case to show well-defined lesions of jaagsiekte and papilliform proliferations with columnar epithelium into lumina of bronchi.
4134	L.R. 7563	8/ 7/24	Amersfoort, Tvl.....	—	Specimen..........	Well-defined jaagsiekte foci, here and there lymphoid infiltration, some jaagsiekte foci confluent.
4451	S. 9963	12/10/24	Bloemfontein.......	13/10/24	1 day at Lab......	Numerous disseminated jaagsiekte foci, many confluent, some with good deal of stroma, in places localized lymphoid infiltration.
4518	S. 7540	20/ 7/23 (6-tooth)	Beaufort West, C.P.	25/11/24	About 16 months at Lab.	Young jaagsiekte foci to those of large dimensions with extensive papilliform formations, not much stroma in the latter, acute pneumonia also present.
4568	S. 9988	10/11/24 (lamb)	Kaffersdam, O.F.S..	16/ 6/27	31 months at Lab..	Definite circumscribed lesions of jaagsiekte, some with good deal of stroma, papilliform formations into lumina of bronchioles, note acute catarrhal pneumonia.
4614	L.R. 7264	29/12/24	Devon, Tv.........	—	Specimen..........	Extensive confluent jaagsiekte foci, with papilliform proliferations, lymphoid infiltration well circumscribed, lung tissue itself not much involved, some proliferations into lumina of bronchioles.
4615	L.R. 7265	29/12/24	Devon, Tvl.........	—	Specimen..........	Extensive confluent jaagsiekte foci, embedded in lung tissue not much changed (the whole resembling glandular organ like the udder).
4622	S. 9985	10/11/24	Taungs, C.P........	7/12/25	About 58 days at Lab.	Jaagsiekte foci with the stroma extensively enlarged. In some sections well-defined jaagsiekte foci present. No lung changes.
4628	S. 10691	20/12/24	Philipstown, C.P....	18/ 1/25	29 days at Lab.....	Early well-defined jaagsiekte foci, to extensive lesions, which themselves are fairly well circumscribed

APPENDIX—(CONTINUED).

(*a*) *Jaagsiekte Cases*—(continued).

Specimen No.	No. of Sheep or No. of Lab. Report.	Date of Arrival.	From Where.	Date of Death and Cause.	Remarks.	Pathological Histology.
4634	S. 10843	20/12/24	Philipstown, C.P....	14/ 1/25	25 days at Lab.....	Few early jaagsiekte foci to extensive foci with a good deal of stroma, here and there lymphoid infiltration. Large jaagsiekte foci also well circumscribed.
4738	S. 11092	14/ 1/25	Philipstown, C.P....	15/ 1/25	1 day at Lab......	Early jaagsiekte foci, to extensive ones with good deal of stroma, embedded in lung, tissue not much changed.
4650	S. 9374	14/ 7/24	Middelburg, C.P.....	19/ 1/25	6 months at Lab...	Do.
4655	L.R. 8548	20/ 1/25	Lichtenburg, Tvl....	—	Specimen..........	Do., plus localized lymphoid infiltration.
4677	S. 10582	19/12/24	Philipstown, C.P....	22/ 1/25	33 days at Lab.....	Extensive jaagsiekte foci and good deal of stroma, lymphoid infiltration, embedded in lung with slight changes. Patches of acute pneumonia.
4679	S. 11035	14/ 1/25	,,	26/ 1/25	12 days at Lab....	Multiple jaagsiekte foci in lung, tissue not much changed.
4686	S. 11103	14/ 1/25	,,	28/ 1/25	14 days at Lab.....	Jaagsiekte lesions with large amount of stroma present.
4708	S. 11090	14/ 1/25	,,	3/ 2/25	20 days at Lab.....	Extensive jaagsiekte foci, with good deal of stroma and lymphoid infiltrations. In another section early jaagsiekte foci seen.
4719	S. 10986	14/ 1/25	,,	4/ 2/25	21 days at Lab.....	Few definite jaagsiekte foci complicated with necrotic foci, probably Preisz-nocard.
4722	L.R. 9567A.	5/ 2/25	?	—	Specimen..........	Jaagsiekte foci and acute catarrhal pneumonia (decomposed).
4736	S. 9984	10/11/24	Taungs, C.P........	9/ 2/25	3 months at Lab...	Well-circumscribed foci of jaagsiekte in portions of lung, not much changed.
4770	L.R. 730	26/ 2/25	Lichtenburg, Tvl....	—	Specimen.........	Extensive confluent jaagsiekte foci (like mamma).
4882	S. 9483	—	—	Died, 9/4/25, arsenical poisoning.	—	Few (about three) jaagsiekte foci in lung, not much changed. In another section some of foci show good deal of stroma. One portion of lung with acute catarrhal pneumonia.
4905	—	—	Abattoirs, Pretoria..	22/ 4/25	Specimen..........	Extensive jaagsiekte foci with good deal of stroma, lymphoid infiltrations.

4930	S. 11666	20/ 3/25	Philipstown, C.P....	3/ 5/25	1½ months at Lab...	Well-defined jaagsiekte lesions and acute pneumonia.
5095	S. 11716	?	?	24/ 6/25	—	Well-defined jaagsiekte foci, many with good deal of stroma, acute pneumonia
5259	S. 5013	7/ 7/22	Bestersput, O.F.S...	23/ 7/25	3 years at Lab......	Few jaagsiekte foci and acute pneumonia.
5274	L.R. 8198	28/ 7/25	Harrismith, O.F.S..	—	Specimen..........	Jaagsiekte foci, many confluent, slight lung changes, few lymphoid infiltrations.
5396	S. 14969	—	—	18/ 9/25	—	Few jaagsiekte foci and acute pneumonia.
5475	S. 9155	9/ 7/24	Middelburg, C.P.....	8/ 6/25	13 months at Lab...	Do.
5533	S. 11117	14/ 1/25	Philipstown, C.P....	Died, 31/10/25	9 months at Lab...	Well-defined jaagsiekte foci with acute pneumonia in whole section.
5769	L.R. 9396	22/ 2/26	Douglas, C.P.......	—	Specimen..........	Early well-circumscribed jaagsiekte foci and acute pneumonia.
5790	L.R. 9790	1/ 3/26	Carnarvon, C.P.....	—	Specimen..........	Foci of jaagsiekte with papilliform proliferations, but not well defined, good deal of stroma, lymphoid infiltrations.
5795	L.R. 30	3/ 3/26	Douglas, C.P........	—	Specimen..........	Early foci of jaagsiekte with extensive acute pneumonia, also large jaagsiekte foci with good deal of stroma.
5812	S. 14312	14/ 1/26	Philipstown, C.P....	10/ 3/26	2 months at Lab....	Diffuse confluent foci and good deal of stroma and acute pneumonia, in some sections few jaagsiekte foci in normal lung.
5841	S. 14497	26/ 3/26	Ladybrand, O.F.S...	27/ 3/26	1 day at Lab......	Well-defined jaagsiekte foci, some with good deal of stroma.
5844	S. 14498	26/ 3/26	,,	1/4/26	6 days at Lab......	Atypical jaagsiekte foci with extensive induration of the lung tissue.
5915	S. 12975	31/ 8/25	Philipstown, C.P....	Died, sequel dipping, 2/5/26	9 months at Lab.....	One jaagsiekte focus, rest acute pneumonia. In a second section jaagsiekte foci more pronounced, note different dimensions of epithelium in connection with proliferations. Some foci with good deal of stroma.
5962	L.R. 5561	1/ 6/26	Steynsrust, O.F.S...	—	Specimen..........	Early jaagsiekte lesions in normal lung, some foci with good deal of stroma and columnar epithelium.
6011	—	18/ 6/26	Johannesburg Abatoirs	—	Specimen..........	Well-defined jaagsiekte foci, with good deal of stroma.
6176	S. 14768	17/ 8/26	De Aar, C.P........	Dead on arrival	—	Jaagsiekte foci with good deal of stroma in acute catarrhal pneumonia.
6224	S. 14976	2/ 9/26	Waterberg, Tvl.....	3/ 9/26	1 day at Lab......	Well-circumscribed jaagsiekte foci in normal lung, some with columnar epithelium.
6281	S. 14627	26/ 6/26	Umtata, Transkei...	21/ 9/26	3 months at Lab...	Few well-circumscribed jaagsiekte foci with good deal of stroma and pneumonia in places. Lymphoid tissue with follicles associated with some bronchioles.
6430	S. 15754	17/12/26	Philipstown, C.P....	Died, 18/12/26	1 day at Lab......	Well-circumscribed jaagsiekte foci, some with good deal of stroma. Here and there acute pneumonia.

APPENDIX—(CONTINUED).

(*a*) *Jaagsiekte Cases*—(continued).

Specimen No.	No. of Sheep or No. of Lab. Report.	Date of Arrival.	From Where.	Date of Death and Cause.	Remarks.	Pathological Histology.
6440	S. 15005	8/ 9/26	Philipstown, C.P....	22/12/26	3 months at lab...	Multiple early jaagsiekte foci, little pneumonia, some foci with good deal of stroma, some foci confluent.
6552	L.R. 12473A.	3/ 2/27	Tweespruit.........	—	Specimen..........	Extensive jaagsiekte foci with good deal of stroma.
6668	S. 10641	19/12/24	Philipstown, C.P....	Killed. 4/3/27	27 months at Lab...	Well-defined jaagsiekte foci, some with good deal of stroma, in places multiple well-formed lymphoid nodules.
6901	S. 7405	20/ 7/23	Beaufort West, C.P.	Died, 22/5/27, uraemic poisoning	46 months at Lab..	Very extensive and advanced induration of the lung tissue. In one section definite papilliform proliferations, like jaagsiekte foci.
6927	L.R. 19260	30/ 5/27	Cradock, C.P.......	—	Specimen..........	Well-defined early jaagsiekte foci in normal lung.
6962	L.R. 11090	9/ 6/27	Tweespruit, O.F.S...	—	Specimen..........	Well-defined jaagsiekte foci, some with good deal of stroma. in places proliferations with columnar epithelium; in places acute pneumonia; in places induration of lung.
6963	L.R. 11091	9/ 6/27	Tweespruit, O.F.S...	—	Specimen..........	Multiple jaagsiekte foci, some with good deal of stroma and acute catarrhal pneumonia, i.e. pneumonia definitely superimposed on jaagsiekte foci.
6969	S. 17149	9/ 6/27	Philipstown, C.P....	15/ 6/27	6 days at Lab......	Lesions of jaagsiekte with good deal of stroma. acute pneumonia.
6974	S. 16344	22/ 3/27	Beaufort West, C.P.	Died, 16/ 6/27	4 months at Lab...	Early jaagsiekte foci, to large foci with good deal of stroma. lung not much changed.
7038	L.R. 14416	30/ 6/27	Postmasburg, O.F.S.	—	Specimen..........	Few well-defined isolated foci of jaagsiekte, papilliform proliferations into bronchioles, in normal lung.
7104	L.R. 15907	7/ 7/27	Tweespruit, O.F.S..	—	Specimen..........	Early jaagsiekte foci to those with a good deal of stroma in normal lung. present only in places.
7215	L.R. 23065	15/ 8/27	,,	—	Specimen..........	Jaagsiekte foci with good deal of stroma plus acute pneumonia.
7230	L.R. 23795	–/ 8/27	,,	—	Specimen..........	Well-circumscribed jaagsiekte foci and acute pneumonia.

7269	S. 18262	22/ 8/27	Burghersdorp, C.P..	Killed, 27/ 8/27	5 days at Lab......	Jaagsiekte foci in normal lung to confluent ones with good deal of stroma. Also induration of lung.
7278	S. 17938	2/ 8/27	Tweespruit, O.F.S...	Died, 29/ 8/27	27 days at Lab.....	Early jaagsiekte foci to those showing extensive proliferations in normal lung.
7292	L.R. 26281	–/ 9/27	,,	—	Specimen..........	Early well-circumscribed foci in normal lung.
7324	L.R. 27125	9/ 9/27	,,	—	Specimen..........	Few jaagsiekte foci in acute pneumonia.
7325	L.R. 27126	9/ 9/27	,,	—	Specimen..........	Do.
7326	L.R. 27126	9/ 9/27	,,	—	Specimen..........	Do.
7340	L.R. 28053	18/ 9/27	,,	—	Specimen..........	Jaagsiekte foci and acute pneumonia.
7355	L.R. 29292	19/ 9/27	,,	—	Specimen..........	Early jaagsiekte foci in acute pneumonia.
7390	S. 18373	20/ 9/27	,,	Died, 1/10/27	12 days at Lab.....	Young jaagsiekte foci to older ones with certain amount of stroma, lung not much altered.
7394	S. 18372	20/ 9/27 (2½ months old)	,,	Died, 4/10/27	14 days at Lab.....	Early jaagsiekte lesions to those with good deal of stroma, with no lung changes. In another section acute pneumonia.
7431	S. 17434	16/ 6/27	Aliwal North, C.P...	Died, 19/10/27	4 months at Lab....	Early well-circumscribed jaagsiekte foci to older ones in normal lung.
7467	L.R. 36311	–/10/27	Rosendal, O.F.S....	—	Specimen..........	From early jaagsiekte foci to those with good deal of stroma.
7489	S. 18339	26/ 8/27	Tweespruit, O.F.S...	Killed, 5/11/27, *in extremis*	3 months at Lab...	Early jaagsiekte foci in normal lung.
7503	S. 18371	20/ 9/27	,,	Died, 12/11/27	2 months at Lab...	From one jaagsiekte focus in normal lung to many in normal lung.
7509	S. 18590	11/11/27	,,	Died, 13/11/27	2 days at Lab......	Well-defined lesions of jaagsiekte (decomposed).
7515	L.R. 41089	15/11/27	,,	—	Specimen..........	Well-defined foci to advanced lesions with large amount of stroma, in acute catarrhal pneumonia.
7606	L.R. 46258	13/12/27	Tweespruit, O.F.S...	—	Specimen..........	Multiple well-defined lesions of jaagsiekte, some with good deal of stroma, acute pneumonia.
7699	L.R. 51749	4/ 2/28	,,	—	Specimen..........	Early lesions of jaagsiekte, some with good deal of stroma, in acute pneumonia.
7700	L.R. 51750	4/ 2/28	,,	—	Specimen..........	Well-defined jaagsiekte foci and acute pneumonia.
7701	L.R. 51751	4/ 2/28	,,	—	Specimen..........	Well-defined jaagsiekte foci and acute pneumonia.
7702	L.R. 51752	4/ 2/28	,,	—	Specimen..........	Well-defined jaagsiekte foci and acute pneumonia.
7753	S. 19482	23/ 1/28	Heilbron, O.F.S.....	Killed, 18/ 2/28	Bought from Luckhoff, Graaff-Reinet	Well-circumscribed jaagsiekte foci in extensive lung induration.
7781	S. 18340	26/ 8/27	Tweespruit, O.F.S...	Died, 26/ 2/28	6 months at Lab...	Multiple jaagsiekte foci to confluent ones and induration of lung.
7799	L.R. 35550	2/ 3/28	,,	—	Specimen..........	Jaagsiekte foci, some with good deal of stroma and acute pneumonia.

APPENDIX—(CONTINUED).

(*a*) *Jaagsiekte Cases*—(continued).

Specimen	No. of Sheep or No. of Lab. Report.	Date of Arrival.	From Where.	Date of Death and Cause.	Remarks.	Pathological Histology.
7817	S. 18592	11/11/27	,,	Killed, 7/ 3/28	4 months at Lab...	Few well-circumscribed jaagsiekte foci in normal lung.
7845	S. 19607	1/ 3/28	,,	Died, 18/ 3/28	17 days at Lab.....	Early jaagsiekte foci to large confluent ones and acute pneumonia.
7927	S. 19608	3/ 3/28	,,	Died, 5/ 4/28	1 month at Lab....	Jaagsiekte foci in acute pneumonia.
7949	L.R. 2009	–/ 4/28	,,	—	Specimen..........	Jaagsiekte foci with good deal of stroma.
7960	L.R. 2525	23/ 4/28	,,	—	Specimen..........	From early foci in normal lung in some sections, to confluent ones with good deal of stroma.
7972	L.R. 2939	24/ 4/28	,,	—	Specimen..........	Well-circumscribed jaagsiekte foci with slight catarrhal pneumonia, some foci with good deal of stroma.
7992	L.R. 3090	26/ 4/28	,,	—	Specimen..........	Do.
8009	L.R. 4162	2/ 5/28	,,	—	Specimen..........	Old jaagsiekte foci.
8013	L.R. 4389	4/ 5/28	,,	—	Specimen..........	Jaagsiekte foci in acute pneumonia.
8036	S. 19647	2/ 5/28	,,	14/ 5/28	12 days at Lab.....	Early jaagsiekte foci in acute pneumonia.
8044	L.R. 6554	–/ 5/28	Vryburg, C.P.......	—	Specimen..........	Early jaagsiekte foci in normal lung to well-formed ones with good deal of stroma.
8086	L.R. 9097	6/ 6/28	Vredefort. O.F.S....	—	Specimen..........	Early jaagsiekte foci in acute pneumonia.
8109	L.R. 10042	13/ 6/28	Tweespruit.........	—	Specimen..........	A few isolated jaagsiekte foci and acute pneumonia.
8185	L.R. 14064	6/ 7/28	Ladybrand, O.F.S...	—	Specimen..........	From early jaagsiekte foci to ones with good deal of stroma.
8241	S. 19646	2/ 5/28	Tweespruit.........	Died, 23/ 7/28	2 months at Lab...	Early jaagsiekte foci in normal lung.
8249	S. 16094	3/ 2/27	Nooitgedacht, Tvl...	Died, 25/ 7/28	17 months at Lab..	Well-circumscribed jaagsiekte foci, some with good deal of stroma.
8287	L.R. 19222	7/ 8/28	Graaff-Reinet, C.P..	Sheep killed	Specimen..........	Jaagsiekte foci, some with good deal of stroma in normal lung, papilliform formations into bronchioles.
8288	L.R. 19223	7/ 8/28	Tweespruit.........	—	Specimen..........	From early jaagsiekte foci in normal lung.

8290	S. 21395	7/ 8/28	,, ...	Killed, 8/ 8/28	1 day at Lab......	Multiple jaagsiekte foci, from early lesions in normal lung to confluent ones.
8362	S. 17340	15/ 6/27	Philipstown, C.P....	Died, sequel dipping, 25/ 8/28	2 months at Lab...	Jaagsiekte foci in all stages, and acute pneumonia.
8390	L.R. 26511	8/ 9/28	Tweespruit.........	—	Specimen..........	Well-circumscribed jaagsiekte foci in acute pneumonia.
8409	L.R. 27229	12/ 9/28	Graaff-Reinet.......	—	Specimen..........	From early jaagsiekte foci in normal lung to some with many papilliform formations and good deal of stroma.
8440	L.R. 28525	19/ 9/28	Tweespruit.........	—	Specimen..........	Well-defined jaagsiekte foci in all stages, in acute pneumonia.
8485	S. 19643	30/ 4/28	Petrusburg, C.P....	Died, 27/ 9/28	5 months at Lab...	From early jaagsiekte foci to well-formed nodules in normal lung.
8532	L.R. 34740	9/10/28	Kinross, Tvl........	—	Specimen..........	From early jaagsiekte foci to well-formed ones in normal lung.
8562	L.R. 37433	18/10/28	Tweespruit.........	—	Specimen..........	Early jaagsiekte foci in normal lung to well-formed ones in acute pneumonia.
8645	S. 21840	25/10/28	Kinross, Tvl........	Died. 21/11/28	28 days at Lab.....	From early jaagsiekte foci in normal lung to well-formed ones which are confluent.
8764	L.R. 56786	27/12/28	Graaff-Reinet.......	—	Specimen..........	Do.
8750	S. 14534	Jaagsiekte Exp. S. 3208	Lab. Sheep at Tweespruit.	Killed, Abattoirs, Pretoria, 20/12/28	—	Well-circumscribed jaagsiekte foci, some with good deal of stroma and lymphoid infiltrations here and there. Lung tissue well defined.
8796	S. 15699	,,	,,	,,	—	A localized Preisz-nocard infection together with a few well-defined jaagsiekte foci.
8823	S. 14524	Jaagsiekte Exp. S. 3156	Laboratory	Killed, Abattoirs, Pretoria, 3/1/28	—	Only one jaagsiekte focus detected, papilliform proliferations with a fair amount of stroma, a few bronchioles, however, show papilliform formations into the lumina.
4480	S. 9876	24/ 9/24	—	30/ 9/24	6 days at Lab......	Multiple lymphoid follicles in lung.
4853	S. 11638	16/ 3/25	Krugersdorp.......	31/ 3/25	15 days at Lab.....	Extensive induration of lungs with proliferations of epithelium, of the nature of loops, in the new connective tissue. Also lymphoid infiltrations here and there. In places nature of a chronic peribronchitis.
5759	File 12/20/1926	—	Montana, U.S.A....	—	—	Distinct but irregular proliferations of epithelium, not adenoma-like, but associated with a chronic indurative pneumonia. Note the columnar nature of the epithelium of these proliferations throughout, and note the eosinophilic infiltration in the stroma. These proliferations do not occur regularly throughout the section as in jaagsiekte. Lymphoid infiltrations in places.

APPENDIX—(CONTINUED).

(*a*) *Jaagsiekte Cases*—(continued).

Specimen No.	No. of Sheep or No of Lab. Report.	Date of Arrival.	From Where.	Date of Death and Cause	Remarks.	Pathological Histology.
7488	L.R. 37677	5/11/27	Tweespruit.........	—	Specimen..........	Peribronchitis and peribronchiolitis.
7516	L.R. 41090	15/11/27	,, ...	—	Specimen..........	Do.
8091	L.R. 9733	8/6/28	,, ...	—	Specimen..........	Proliferations of bronchiolar epithelium, not jaagsiekte. Note also collapse of lung in places.
8286	L.R. 19222	7/8/28	Graaff-Reinet Exp. Station, C.P.	—	Specimens from 3 sheep all similar in appearance	Extensive lymphoid hyperplasia in some sections, some not associated with bronchioles. In other sections apparent proliferation of bronchiolar (columnar) epithelium with collapse of the lung tissue. No lesions of typical jaagsiekte.
8298	S. 16703	17/5/27	Laboratory.........	Died, 7/8/28, tar expt.	—	Note few lymphoid nodules associated with bronchioles.
8387	S. 19642	30/4/28	Bloemfontein, O.F.S.	Died, 6/9/28	About 5 months at Lab.	Chronic catarrhal pneumonia with lymphoid infiltrations and the presence of foreign body giant cells in the exudates in the lumina of alveoli.
8419	S. 19640	30/4/28	Petrusburg.........	Died, 13/9/28	—	Extensive induration of lung with epithelial proliferations, in the form of loops, not like jaagsiekte, but like those associated with a chronic inflammation, also lymphoid infiltrations here and there.
8439	L.R. 28534	19/9/28	Graaff-Reinet Expt. Station, C.P.	—	Specimen..........	Catarrhal pneumonia, here and there with slight induration and lymphoid infiltrations.
8527	S. 21645	7/9/28	,,	Died, 5/10/28	—	Acute catarrhal pneumonia.
8537	S. 21647	7/9/28	,,	9/10/28	—	Extensive induration with a good deal of lymphocytes in the granulation tissue, disseminated irregular epithelial proliferations probably associated with a chronic pneumonia of the lungs.

8621	S. 21806	—	—	14/11/28	Lung worm expt....	Catarrhal pneumonia with indurative changes here and there.
8665	S. 21646	7/9/28	Graaff-Reinet Expt. Station, C.P.	Died, 29/11/28	—	Same as for 8537.
8765	L.R. 50787	—	,,	—	Specimen..........	Slight increase of connective tissue in the septa.
8797	S. 15977	—	Jaagsiekte Exp. S. 3208	Killed, Abattoirs, Pretoria, 3/1/29	—	Bronchioles with broadened epithelium and their walls infiltrated with lymphoid cells. Places of collapse. In places no changes in lung, and in others granulation tissue with Langhanse-like giant cells

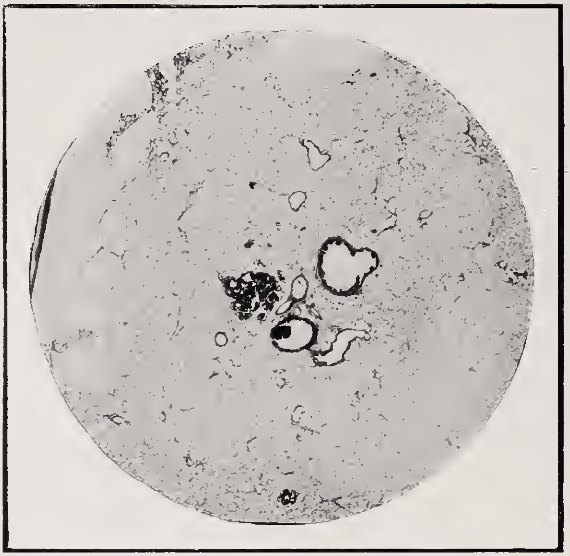

FIG. 1.

Specimen No. 7038. Lung of sheep. Jaagsiekte. (Magnified 7 ×.) Several young disseminated Jaagsiekte foci, and one well-formed lesion. Note also papilliform proliferation into a bronchiole. Rest of the lung substance shows practically no changes.

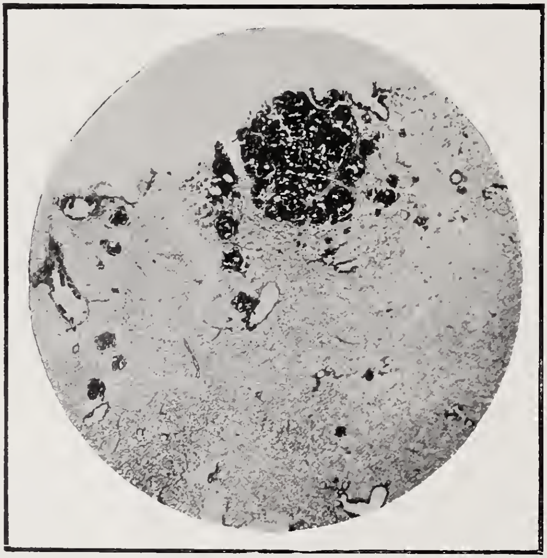

FIG. 2.

Specimen No. 7038. Lung of sheep. Jaagsiekte. (Magnified 7 ×.) Several young disseminated Jaagsiekte foci, and one well-formed lesion.

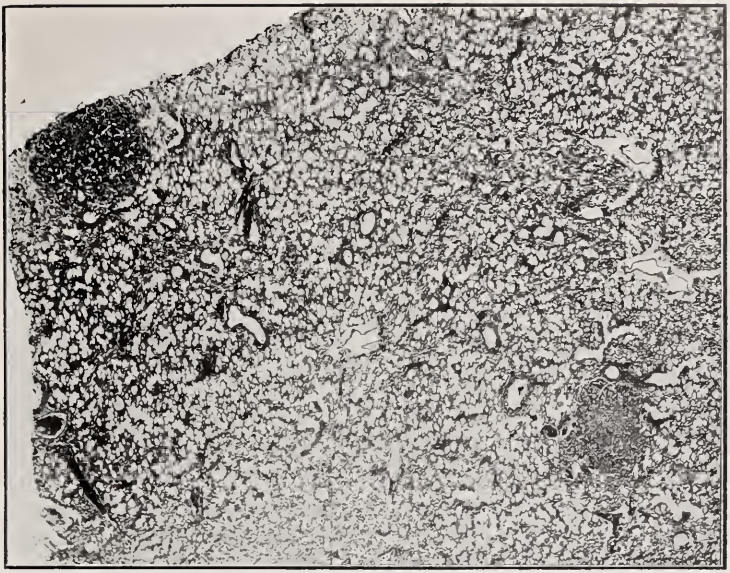

Fig. 3.
Specimen No. 7583. Lung of sheep. Jaagsiekte. (Magnified 20 ×.) Note the presence of two well-formed adenomata in more or less normal lung.

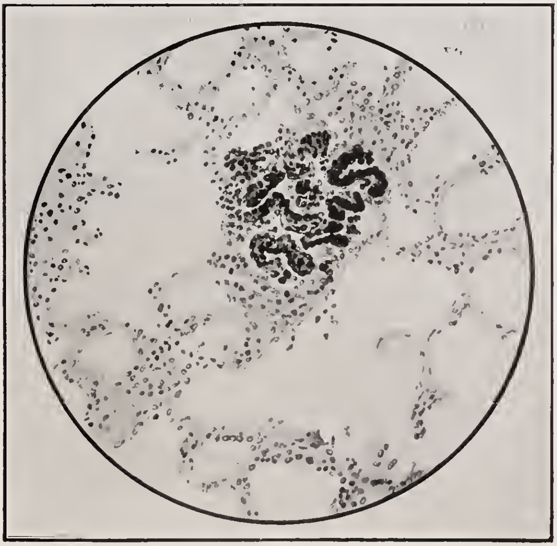

Fig. 4.
Specimen No. 7431. Lung of sheep. Jaagsiekte. (Magnified 170 ×.) Shows a well-formed proliferation in an early Jaagsiekte focus.

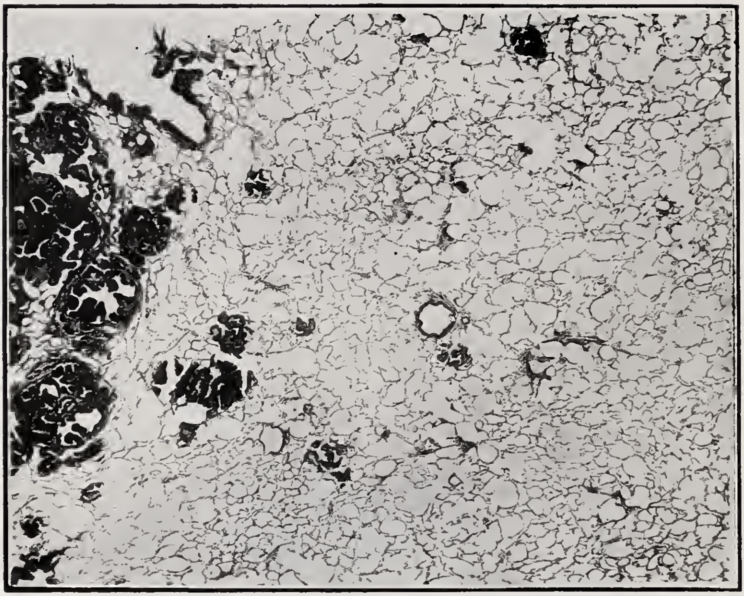

Fig. 5.

Specimen No. 7038. Lung of sheep. Jaagsiekte. (Magnified 30 ×.) Note early lesions of Jaagsiekte, to those in which the papilliform proliferations are well developed. The rest of the lung tissue practically normal.

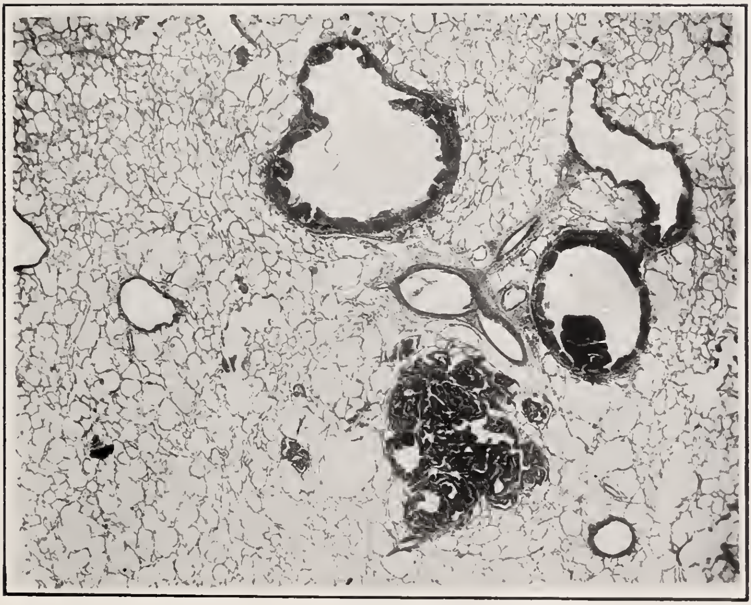

FIG. 6.

Specimen No. 7038. Lung of sheep. Jaagsiekte. (Magnified slightly higher than 30 ×.) Note presence of a papillomatous proliferation into the lumen of a bronchiole.

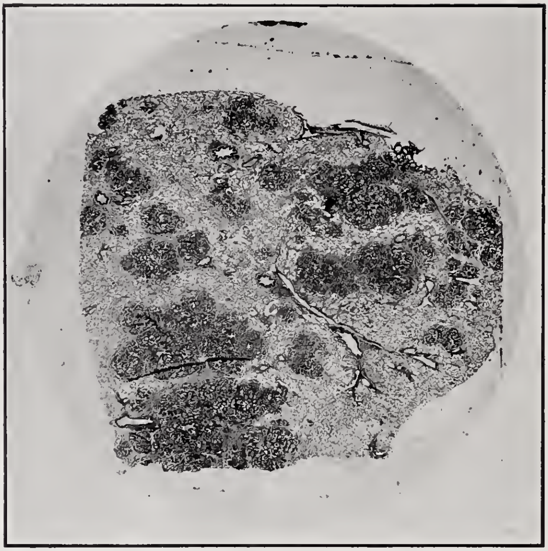

Fig. 7.

Specimen No. 4736. Lung of sheep. Jaagsiekte. (Magnified 7 ×.) Note numerous Jaagsiekte foci. Some have become confluent. The presence of the papilliform proliferation is fairly characteristic. The remaining portion of the lung substance shows very slight alterations.

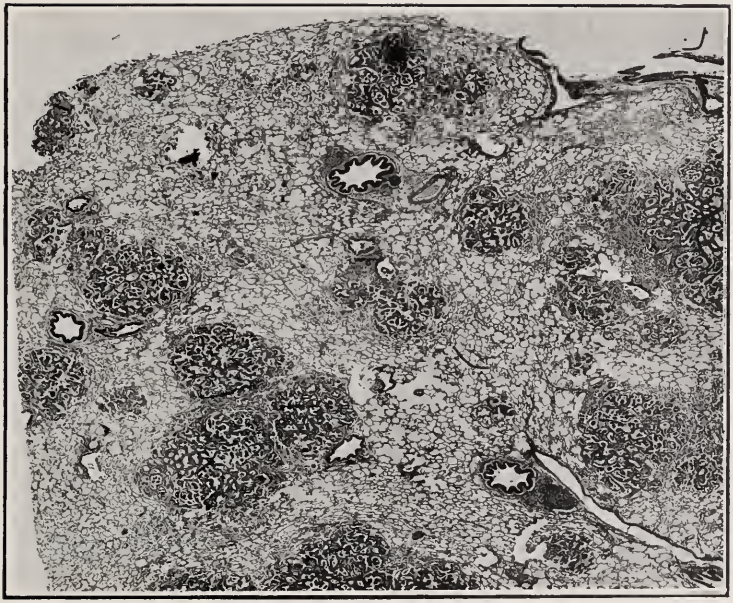

FIG. 8.

Specimen No. 4736. Lung of sheep. Jaagsiekte. (Magnified 17 ×.) Note numerous Jaagsiekte foci; some have become confluent. The presence of the papilliform proliferation is fairly characteristic. The remaining portion of the lung substance shows very slight alterations.

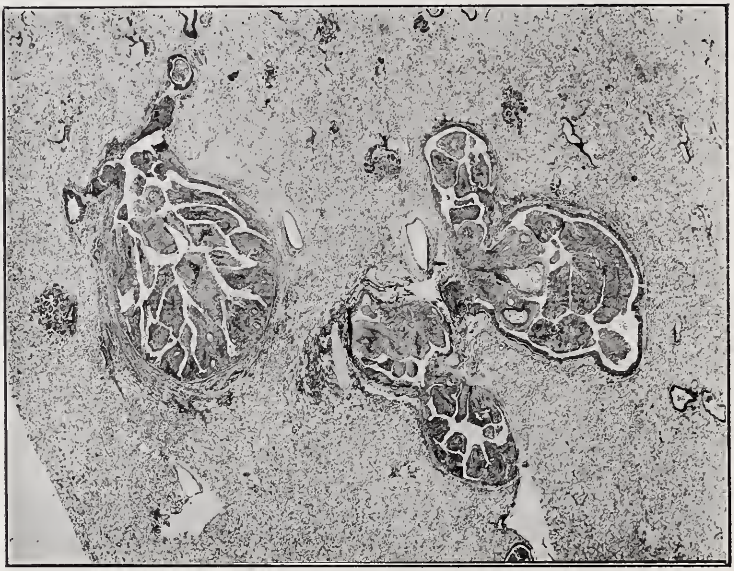

FIG. 9.

Specimen No. 3432. Lung of sheep (magnified 20 ×). Numerous isolated very early Jaagsiekte foci, associated with the alveolar epithelium. Proliferations formed in connection; several bronchi also present in this section.

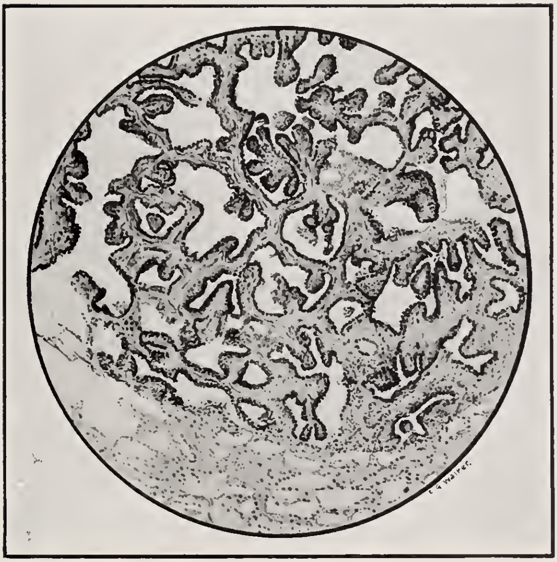

FIG. 10.

Specimen No. 7972. Lung of sheep. Jaagsiekte. (Photograph of a coloured drawing.) Note characteristic papilliform formation in a well-formed Jaagsiekte focus. The surrounding lung tissue shows practically no changes. (Magnified 90 ×.)

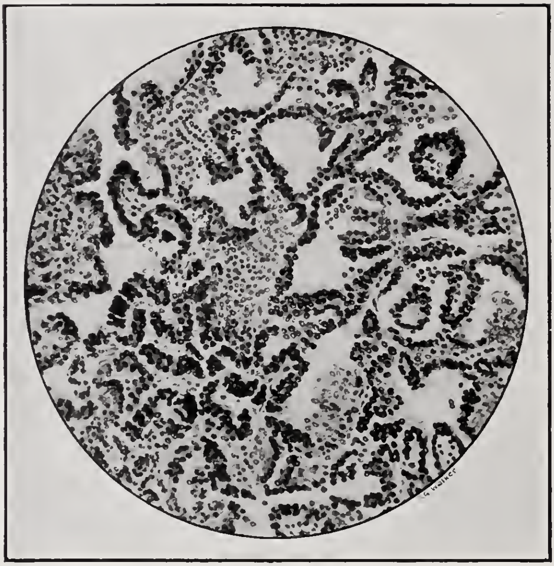

Fig. 11.

Specimen No. 8485. Lung of sheep. Jaagsiekte. (Photograph of coloured drawing.) Note the papilliform proliferations and in places a good deal of stroma. (Magnified 136 ×.)

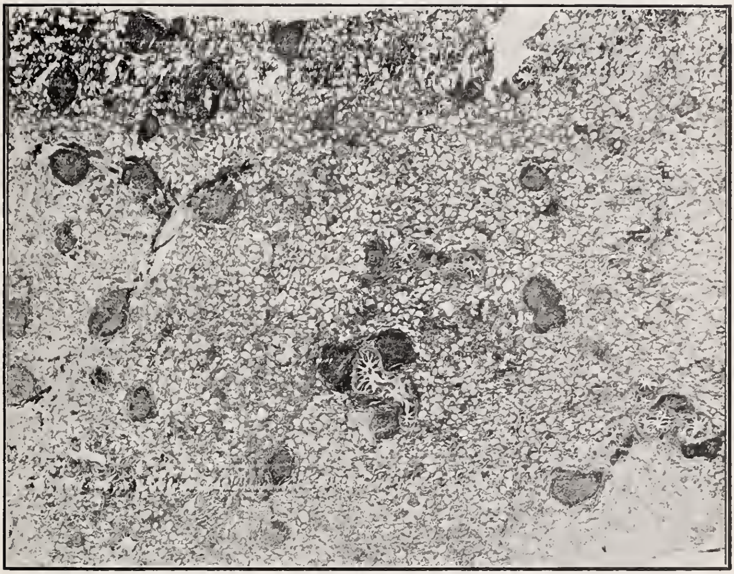

FIG. 12.

Specimen No. 8286. Lung of sheep, Graaff-Reinet Experimental Station. (Magnified 30 ×.) Multiple disseminated lymphoid follicles. Some closely associated with bronchioles. Note the presence of "germinal centres." Rest of the lung tissue shows very few changes.

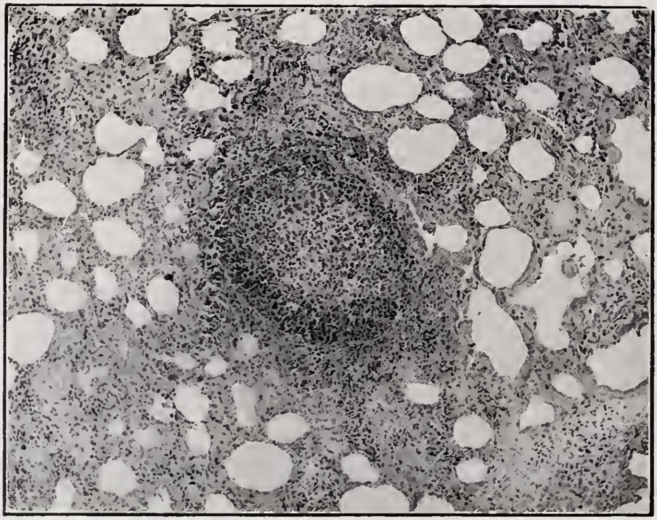

Fig. 13.

Specimen No. 8286. Lung of sheep, Graaff-Reinet Experimental Station. (Magnified 100 ×.) Note the presence of a well-formed lymphoid nodule in the lung substance.

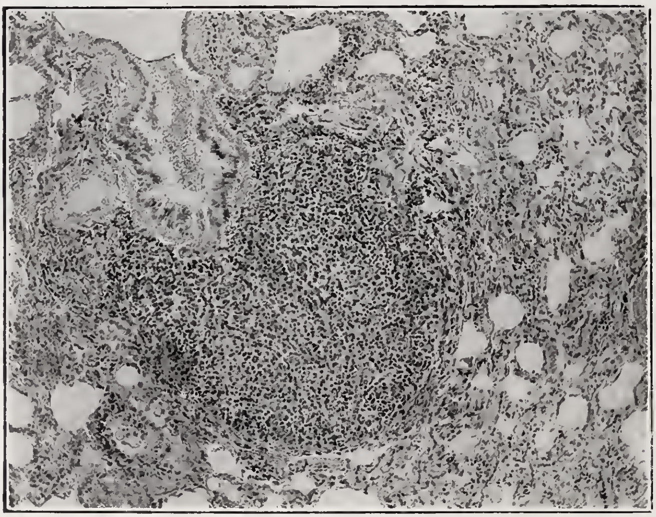

Fig. 14.

Specimen No. 8286. Lung of sheep, Graaff-Reinet Experimental Station. (Magnified 100 ×.) Note the presence of a well-formed lymphoid nodule in the lung substance.

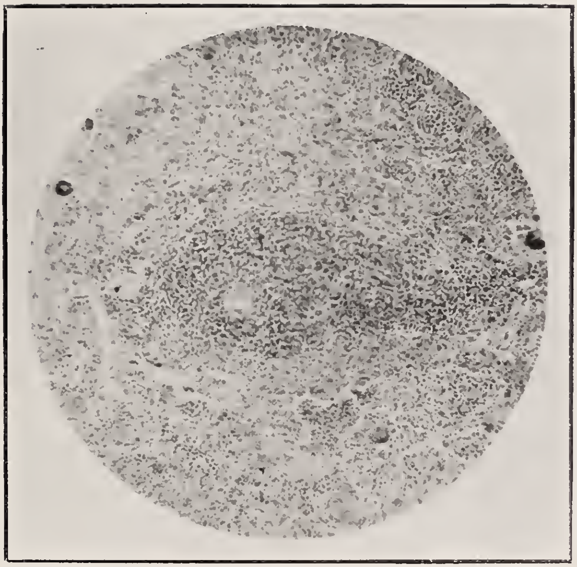

FIG. 15.

Specimen No. 8387. Lung of sheep (magnified 100 ×). Chronic catarrhal pneumonia with multiple lymphoid infiltrations, and the formation of foreign body giant cells in the exudates present in the lumina.

15th Annual Report of the Director of Veterinary Services, Union of South Africa. October, 1929.

Pathology of Phenol Poisoning in Sheep induced by certain Dips.

By GILLES DE KOCK, D.Sc., Dr.Med.Vet., M.R.C.V.S., Sub-Director of Veterinary Services, Onderstepoort.

THE symptomatology and certain aspects of the etiology of phenol poisoning in sheep induced by certain dips have been discussed by Steyn (1929). It was shown that on a certain farm a percentage of sheep immersed in certain proprietary phenol dips succumbed to phenol poisoning, and in a batch of 252 sheep dipped at the Laboratory on the 10/9/28, 56 animals died. Experiments (*vide* S. 3637 and S. 3639) were subsequently carried out to show that poisoning did ont result from ingestion of the dip, but that absorption had taken place through the intact skin without provoking any lesions in the latter. Acute symptoms were observed in some within 24 hours and they were mainly characterized by a marked dyspnoea and respiratory distress. A number of sheep died within 48 hours of dipping, while the others lingered for three and four days.

PATHOLOGICAL ANATOMY.

A description of the pathological changes seen in the experimental cases will be found in the Appendix. All carcases were characterized by marked cyanosis, especially evident in the subcutaneous connective tissues. The inner layer of the skin was reddish blue in appearance, and showed a rich arborescence of minute blood-vessels. In the majority of cases the phenol smell could easily be detected when the carcase was opened; especially was this noted in the caecum. Serous cavities showed a quantity of transudate, which in some cases was abundant, especially in the thorax and pericardium. In practically all cases a certain amount of emphysema was found. Sometimes this was very extensive and involved many tissues over a considerable area. In some cases emphysema occurred in the lungs, mediastinum, subcostal pleura peritoneum and peritracheal tissues in the cervical region, from where it extended into the subcutis. In a number of cases the lungs revealed large subpleural air vesicles, which often reached the size of an orange and even larger. Another very characteristic feature was the presence of glottis oedema. Sometimes unilateral oedema was present in the folds of the larynx, and in other cases the pharynx and trachea were also involved.

The mucosa of the upper respiratory tract usually revealed a hyperaemia, which in some instances was intense and complicated with haemorrhages. In a few cases a slight acute catarrhal inflammation of the mucosa was evident.

The lungs in all cases were extensively affected. They varied in colour from a light bluish red to an intense dark bluish red. No consolidations were identified, but the lungs as a whole presented a characteristic firm appearance. It was much less resilient and elastic. On section a good deal of blood of a bluish tinge escaped from the cut surface. This was usually mixed with a certain amount of blood-stained foam. In some instances there were small haemorrhages present in the pleura and in the substance of the lung. The mucosa of the bronchi varied from a light to an intense bluish red, with multiple small haemorrhages. The venous hyperaemia was general throughout the body, and was prominent in such organs as the liver and kidneys. The latter also revealed varying degrees of degenerative changes.

The alimentary tract in some cases was hyperaemic and small haemorrhages were present, but as a rule no changes were observed. Subepicardial and subendocardial haemorrhages were also noted. A number of the lymphatic glands were hyperaemic, and a slight tumour splenis was present in a few cases. Hyperaemia was also detected in the meninges and substance of the central nervous system.

PATHOLOGICAL HISTOLOGY.

Hyperaemia was identified in the skin, liver, kidneys, lymphatic glands, and myocardium. In some cases fatty changes were observed in the liver, myocardium, and kidneys; but rarely were these extensively present in the liver and kidneys. In the liver a slight necrobiosis was also identified. The lungs showed very characteristic changes. In a few instances a slight catarrhal pneumonia was present, but generally exudative changes could not be identified. The lungs were characterized by an extensive hyperaemia. All blood-vessels within the lung, especially the capillaries in the walls of the alveoli, were distended with blood. Certain definite changes had also taken place in the epithelium of the alveoli (see figures 1-4).

The epithelium had assumed a cubical contour in the great majority of cases, and it appeared as if the whole lining of cells of the alveoli had become partly dislocated or shed from the connective tissue overlying the swollen and tortuous capillaries. The epithelial lining in the lumen of an alveolus resembled a shrunken acinus. It may be stated that the nuclei of these cubical-shaped cells stained fairly well. Between this partly shed epithelial lining and the much-distended capillaries there were irregular spaces *between* torn connective tissue fibres. It gave one the impression that something had accumulated between the epithelial layers and the connective tissue layer, and that this was to a certain extent responsible for the shedding of the lining into the lumen of the alveolus. In all sections examined these spaces were clear and nothing of a fluid character or otherwise could be identified in them. Gas had probably accumulated there and had exerted an abnormal pressure on the tissues concerned. That may explain the origin of the emphysema which was noticed in the lungs and various parts of the body.

This picture in the lungs of sheep closely resembles the early lesions described by M'Fadyean (1920) in case of verminous pneumonia in sheep. He says that a remarkable feature frequently observed in parts of lobules showing the purely catarrhal type of lesions was the apparent completeness of the process of desquamation. In some alveoli not a single epithelial cell remained in connection with the wall . . . then there were alterations of special interest, namely, those which entirely transformed the appearance of the oveolar epithelium and thereby the aspect of the whole lung . . . alveolar epithelium in a small group had been retained or renewed, and the cells, instead of having the normal squamous or endothelial form, had assumed a plump cubical shape . . . (these transformations are very characteristic in some of the plates shown by M'Fadyean) . . . the alveolar walls were for the most part denuded of epithelium, but in three or four air-cells there was an incomplete lining of cubical cells, and in another portion of the lung the air-cells and infundibula had acquired a complete lining of similar cells.

In the literature at the disposal of the writer very little information could be found about changes in the respiratory tract after phenol poisoning. Ellinger (1923) refers to the frequent occurrence of inflammatory changes, e.g. tracheo-bronchitis, bronchopneumonia. Wachholz, quoted by Ellinger, obtained bronchopneumonic processes in case of a cat after subcutaneous inoculation and referred to the excretion of phenol by the lungs.

The outstanding features in the above instance of phenol poisoning induced by dipping were: (1) the absorption of phenol through the intact skin, (2) the marked dyspnoea and respiratory distress, and (3) the characteristic changes observed in connection with the alveolar epithelium. Does the lung play a part in the elimination of the phenol absorbed from the skin, and does the respiratory epithelium become damaged in this process? Apparently the major portion of the absorbed phenol is brought to the lungs in the venous blood, i.e. before it reaches such organs as the myocardium, liver, and kidneys. In the majority of cases these latter parenchymatous organs were only damaged to a very slight extent. The damaged alveolar epithelium evidently markedly interferes with the normal interchange of the gases and leads to the extensive emphysema and venous hyperaemia in the lung and rest of the animal body. Fried, in his discussion of the defensive and metabolic apparatus of the lungs, refers to the statement of Roger, viz., that it has also been claimed that alkaloids and various other toxic substances are oxidized in the pulmonary parenchyma. He refers to recent investigations by German workers to the effect that the lungs are extremely sensitive to various substances introduced by the mouth or by way of the blood-stream. In respect of the study of the blood, the writer did not observe any alterations.

CONCLUSIONS.

(1) Phenol poisoning in sheep induced by certain proprietary dips was characterized at post-mortem by a general venous hyperaemia, hydropericardium, hydrothorax, emphysema in lungs,

mediastinum, peritracheal tissues of cervical region, subcutis, oedema of mucosa and larynx, sometimes of pharynx and trachea; hyperaemia of lungs and mucosa of bronchi; sometimes this was complicated with small haemorrhages.

(2) Besides hyperaemic and degenerative changes in certain parenchymatous organs, the lungs revealed characteristic changes microscopically, viz., extensive hyperaemia and cubical transformation of the epithelial lining of he alveoli, which was in part shed, probably as a result of the accumulation of gas between epithelium and connective tissue.

(3) The question as to whether alveolar epithelium plays a part in the elimination of phenol is discussed.

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STEYN, D. G. (1929). "A Note on the Symptomatology of Phenol Poisoning in Sheep induced by certain Dips." (See this Report, pp. 657-658.)

APPENDIX.

No of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
16753	10.9.28	12.9.28	Marked cyanosis visible mucous membranes, slight hydropericard, hydrothorax, hydroperitoneum, slight oedema and hyperaemia lungs, slight tumour splenis, P.M. changes liver and kidneys, degeneration of myocard.
19579	10.9.28	12.9.28	Strong carbolic smell on skin, degenerative changes heart. Venous congestion of mucous membranes, oedema and hyperaemia lungs, bronchitis (?), hyperaemia kidneys (passive ?), glottis oedema; oedema of mucosa of trachea, submucosal haemorrhages. Fatty degeneration and passive hyperaemia liver. (Specimen No. 8412.)
19588	10.9.28	12.9.28	Odour of carbolic acid from internal organs, extensive hyperaemia and oedema lungs, extensive hyperaemia kidneys, fatty degeneration liver, fairly extensive peritracheal emphysema. (Specimen No. 8411.)
18223	10.9.28	12.9.28	Hyperaemia and oedema lungs (well marked), parasitic nodules intestines, extensive peritracheal emphysema; slight unilateral oedema of right epiglottic fold, fatty changes liver, slight hyperaemia kidneys. (Specimen No. 8413.)
20863	10.9.28	12.9.28	Fairly well-marked hyperaemia and oedema lungs; fatty changes liver; slight hyperaemia kidney; hydropericard. (Specimen No. 8414.)
20435	10.9.28	12.9.28	Well-marked hyperaemia and oedema of lungs, fatty changes liver, slight hyperaemia kidneys.

No. of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
20056	10.9.28	12.9.28	Hyperaemia and oedema lungs, emphysema of mediastinum, echinococcus cyst left lung (calcified), fatty degeneration liver, ecchymosis epicard, slight hydropericard and hydrothorax.
19202	10.9.28	12.9.28	Carbolic smell on skin; cyanosis, slight hydropericard. and hydrothorax, marked hyperaemia and oedema lungs, slight hyperaemia kidneys.
19693	10.9.28	12.9.28	Oedema and hyperaemia lungs; emphysema mediastinum, hyperaemia kidneys, degeneration liver, retropharyngeal glands swollen and haemorrhagic, marked smell of tar caecum, slight catarrh intestines, slight hydropericard.
19726	10.9.28	12.9.28	Subpleural emphysema, emphysema and oedema lungs, marked emphysema mediastinum and along course of trachea, slight hydropericard, intestines distended with gas, degeneration liver, thymus shows haemorrhages, hyperaemia kidneys, congestion of blood-vessels of pancreas; sub-epicardial haemorrhages, oedema larynx and pharynx, trachea contains froth and blood-vessels injected; cyanosis all mucous membranes; few parasitic nodules, hyperaemia and haemorrhages intestines (small and large).
21055	10.9.28	12.9.28	Marked oedema, congestion and emphysema lungs, dilatation of heart, hydropericard, extensive degeneration liver, marked hyperaemia small intestines, slight hyperaemia kidneys; froth in bronchi and trachea; oedema larynx, muscular wall of caecum much relaxed, strong smell of tar in caecal contents.
20431	10.9.28	12.9.28	Gas in intestines, cysticerus tenuicollis, hydrothorax, emphysema lungs, under pleura and over ribs; hyperaemia and fatty changes liver. Slight hyperaemia kidneys marked hydropericard with flocculi, dilatation heart, haemorrhages thymus, oedema of mediastinal glands; few parasitic nodules intestine.
19253	10.9.28	12.9.28	Blood deep dark red; cyanosis mucous membranes; oedema, emphysema, and hyperaemia lungs, emphysema trachea and mediastinum, slight oedema larynx and pharynx, hyperaemia kidneys, parasitic nodules and hyperaemia intestines; tarry smell caecum; precrural glands hyperaemic and oedematous.
15995	10.9.28	12.9.28	Fat necrosis, congestion and hyperaemia lungs, slight hydropericard; subendocardial haemorrhages; marked degeneration liver, gas in intestines, tarry smell caecum, and hyperaemia, nodules and nodular worms intestines; oedema trachea and larynx; cyanosis mucous membranes.
20400	10.9.28	12.9.28	Cysticercus tenuicollis cysts. oedema, hyperaemia, and emphysema lungs; slight hydrothorax; haemorrhages endocard and epicard; emphysema mediastinum and cervical peritracheal tissues; hyperaemia kidneys, parasitic nodules liver and intestines, degeneration liver, tarry smell caecum.
19706	10.9.28	12.9.28	Emphysema and hyperaemia lungs; slight hydrothorax; hydropericard; froth in trachea, oedema larynx and pharynx; retropharyngeal glands oedematous and haemorrhagic; emphysema mediastinal tissues; degeneration liver; degeneration of kidneys; fluid in pelvis; hyperaemia mucous membranes; tarry smell caecum.

No. of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
18675	10.9.28	12.9.28	Emphysema, oedema, hyperaemia, and haemorrhages lungs: hydropericard; emphysema mediastinal tissues, under pleura, over ribs and along trachea, slight oedema larynx and pharynx; precrural glands oedematous; swelling, hyperaemia and fatty degeneration liver; hyperaemia kidneys; slight hyperaemia intestines, tarry smell caecum.
19703	10.9.28	12.9.28	Hyperaemia, oedema, and emphysema lungs; extensive emphysema mediastinal and cervical tissues; slight oedema larynx and pharynx, hydropericard and hydrothorax; sub-epicardial haemorrhages; hyperaemia and degeneration liver, hyperaemia kidneys and intestines; tarry smell caecum.
19294	10.9.28	12.9.28	Cyanosis, extensive emphysema in subcutis over whole body: blood deep dark red: emphysema, oedema, and hyperaemia lungs; hydropericard; slight hydrothorax; parasitic nodules intestines; degenerative changes and hyperaemia liver; parasitic nodules liver; hyperaemia kidneys; emphysema mediastinum and all other tissues; injection of all mucous membranes; large parasitic nodules caecum.
17236	10.9.28	12.9.28	Oedema lungs; fatty degeneration liver; emphysema subcutis; slight hyperaemia and catarrh intestines; tarry smell caecum; degeneration kidneys; slight hydropericard.
20796	10.9.28	12.9.28	Hydropericard; hydrothorax; oedema and hyperaemia lungs; blood extravasations bronchi; degeneration liver and kidneys; marked cyanosis visible mucous membranes; slight tumor splenis.
21124	10.9.28	12.9.28	Oedema lungs; subpleural emphysema; hydropericard; fatty degeneration liver and kidneys; marked carbolic smell from abomasum; catarrh of mucosa of abomasum and intestines; marked tarry smell caecum.
20601	10.9.28	12.9.28	Marked cyanosis mucous membranes; hydrothorax; hydropericard; hydroperitoneum; glottis oedema; hyperaemia liver; slight degeneration kidneys; subpleura emphysema; marked hyperaemia and oedema lungs parasitic nodules intestines.
20202	10.9.28	12.9.28	Slight oedema of glottis folds; oedema and emphysema lungs; fatty degeneration liver and kidneys; marked phenol smell rumen, and tarry smell caecum; catarrh of mucosa of stomach; slight hydropericard.
20267	10.9.28	12.9.28	Marked cyanosis mucous membranes, hydropericard; subepicardial haemorrhages; emphysema lungs; marked degeneration liver; hyperaemia kidneys; parasitic nodules intestines.
No No.	10.9.28	12.9.28	Slight glottis oedema; oedema and emphysema lungs; slight hydropericard; tarry smell caecum; slight catarrh of mucosa of abomasum and small intestines; subpleural emphysema; numerous parasitic nodules intestines.
20364	10.9.28	12.9.28	Hydrothorax; hydropericard; subpleural emphysema; oedema lungs; marked hyperaemia and degeneration liver; marked hyperaemia kidneys.
17306	10.9.28	13.9.28	Adiposity; fat necrosis (*no changes in pancreas*); slight hydropericard; dilatation of both ventricles of heart; fairly well-marked degenerative changes liver; slight tumor splenis; slight hyperaemia and degenerative

No. of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
			changes kidney; hyperaemia (extensive) and oedema lungs; hyperaemia trachea and pharynx; *no changes in intestines:* general cyanosis. (Specimen No. 8421.)
17321	10.9.28	13.9.28	General cyanosis; marked hyperaemia and oedema lungs; echinococcus cysts lungs and liver; degenerative changes liver; slight acute catarrhal enteritis; slight hyperaemia kidneys; hyperaemia trachea; peripharyngeal emphysema; marked hydropericard.
20361	10.9.28	13.9.28	General cyanosis; hyperaemia, emphysema, and oedema lungs; degeneration and hyperaemia liver; haemorrhages and slight hyperaemia bronchi and trachea; calcareous nodules intestines (distended with gas); marked hydropericard; subcutaneous emphysema.
18232	10.9.28	13.9.28	Slight glottis oedema; general cyanosis; extensive hyperaemia and oedema lungs; slight epicardial ecchymoses; slight hyperaemia pancreas; extensive degenerative changes liver; hyperaemia and degeneration kidneys; rumen and intestines distended with gas; calcareous nodules intestines; marked hydropericard.
20741	10.9.28	13.9.28	Subepicardial haemorrhages; hyperaemia kidneys; marked hyperaemia, emphysema, and oedema lungs; hyperaemia larynx, pharynx, and trachea; distension of rumen and intestines with gas; slight hyperaemia abomasum; fair number of wireworms present, slight localized catarrhal enteritis.
19590	10.9.28	13.9.28	P.M. changes present; hyperaemia liver; hyperaemia and oedema lungs; hyperaemia larynx, pharynx, trachea; hyperaemia kidneys; degeneration liver and myocard; distension of rumen with gas; slight wireworm infection abomasum; hyperaemia caecum and tarry smell caecum.
19566	10.9.28	13.9.28	Slight hyperaemia and oedema of glottis; hyperaemia of trachea and pharynx; marked emphysema of mediastinum; slight hydropericard and hydrothorax; extensive hyperaemia and oedema lungs; hyperaemia and extensive degeneration liver; hyperaemia of kidneys; slight hyperaemia duodenum; gas in abomasum.
20458	10.9.28	13.9.28	P.M. changes; marked hyperaemia lungs; hydropericard; dilatation of heart; marked localized oedema pharynx; fatty changes and hyperaemia liver; kidney hyperaemia. (Specimen No. 8423.)
19538	10.9.28	13.9.28	Extensive P.M. changes; slight oedema and hyperaemia lungs; hyperaemia larynx, pharynx, trachea; slight oedema glottis; degeneration kidneys, heart, and liver (marked by P.M.C.); rumen distended with gas; wireworms abomasum; hyperaemia caecum; marked carbolic smell throughout.
19730	10.9.28	13.9.28	General cyanosis; extensive fat necrosis; venous hyperaemia and oedema lungs; hyperaemia trachea; extensive hyperaemia and degenerative changes liver; hyperaemia kidneys; hydropericard; hydrothorax.
19728	10.9.28	13.9.28	Fairly extensive P.M. changes; general cyanosis; hydrothorax; hydropericard; decomposition and degeneration liver; decomposition kidneys; oedema and hyperaemia lungs; hyperaemia oesophagus, trachea, larynx; rumen and intestines filled with gas; localized catarrhal enteritis; slight P.M.C. spleen.

No. of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
20874	10.9.28	13.9.28	General cyanosis; slight oedema and hyperaemia larynx; hyperaemia, oedema, and emphysema lungs; hyperaemia and degeneration kidneys; degeneration and hyperaemia liver; localized haemorrhages spleen; subepicardial haemorrhages; slight degeneration myocard; hydropericard; rumen and intestines distended with gas. Localized slight acute enteritis. (Specimen No. 8418.)
19949	10.9.28	13.9.28	Advanced P.M. changes; hydrothorax; hydropericard; emphysema mediastinum; general cyanosis; hyperaemia and oedema glottis; hyperaemia trachea; hyperaemia and oedema lungs; extensive old abrasions lungs.
19961	10.9.28	13.9.28	P.M. changes; general cyanosis; hyperaemia and oedema lungs; slight hydropericard; dilatation and degeneration heart; hyperaemia and degeneration liver and kidneys; slight hyperaemia bladder.
20450	10.9.28	13.9.28	Very extensive P.M. changes; extensive cyanosis; hyperaemia trachea; oedema and hyperaemia lungs; endocardial haemorrhages; slight echinococcus cyst lungs; calcareous nodules large intestines. (Too decomposed.)
20497	10.9.28	13.9.28	Marked P.M. changes; emphysema thorax; hydropericard and hydrothorax; hyperaemia and oedema lungs; hyperaemia and degenerations liver and kidneys; degeneration myocard; hyperaemia larynx and trachea; rumen distended with gas; slight haemonchosis; hyperaemia caecum.
20475	10.9.28	13.9.28	General cyanosis; hyperaemia and degeneration liver; hyperaemia, oedema and emphysema lungs; hyperaemia kidneys; decomposition spleen; hydropericard; dilatation heart; hyperaemia larynx, pharynx, and trachea; gas in rumen and intestines; slight localized catarrhal enteritis.
16904	10.9.28	13.9.28	Hydropericard; hydrothorax; extensive fat necrosis; emphysema mediastinum and along costal pleura; slight hyperaemia and oedema larynx and trachea; extensive hyperaemia; oedema and emphysema lungs; subepicardial haemorrhages; degeneration myocard; degeneration and hyperaemia liver; hyperaemia pancreas; hyperaemia and degeneration kidney; hydronephrosis and calculi kidneys; slight localized acute enteritis.
No No.	10.9.28	13.9.28	Advanced P.M. changes; hyperaemia and oedema in glottis and marked in lungs; hyperaemia trachea; hydrothorax; hydropericard; marked emphysema mediastinum; wireworms; nodular worms; fat necrosis.
18203	10.9.28	13.9.28	General cyanosis; slight hydrothorax; hydropericard, with plasma coagulum; subepicardial ecchymosis; very slight glottis oedema; emphysema mediastinal connective tissue; hyperaemia, oedema, and emphysema lungs; degeneration liver.
17325	10.9.28	14.9.28	Hydropericard; hydrothorax; degeneration myocard; marked oedema and hyperaemia left lung; hepatization stage of catarrhal pneumonia, right lung; P.M.C. liver and kidneys; slight hyperaemia small intestines; tumor splenis.

No. of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
No No.	10.9.28	14.9.28	Hydropericard; hydrothorax; oedema of perilaryngeal and pharyngeal tissues; catarrhal pneumonia; hyperaemia mucosa bronchi; fatty degeneration liver: degeneration kidneys.
19586	10.9.28	14.9.28	Slight glottis oedema; hydropericard; degeneration myocard; marked hyperaemia and oedema lungs; hyperaemia of mucosa bronchi; fatty degeneration liver; degeneration kidneys; tumor splenis.
20381	10.9.28	14.9.28	Hydrothorax; hydropericard; marked hyperaemia and oedema lungs: degeneration kidneys; fatty degeneration liver; tumor splenis; localized haemorrhagic enteritis small intestines.
19712	10.9.28	14.9.28	Hydrothorax; hydropericard; marked hyperaemia and oedema lungs; fatty degeneration liver and kidneys.
19240	10.9.28	14.9.28	Pneumonia, hyperaemia, and oedema lungs; fatty degeneration liver: acute adenitis mediastinal glands; petechiae epicard. (Specimen No. 8424.)
21160	10.9.28	15.9.28	Marked hyperaemia and oedema lungs; hydropericard; slight hydrothorax; marked fatty changes liver; tumor splenis; acute nephritis; slight hyperaemia stomach.
20416	10.9.28	15.9.28	P.M. changes; hyperaemia lungs; hydrothorax; hydropericard; nodular worms intestines; oedema laryngeal region; hyperaemia small intestines.
20375	10.9.28	15.9.28	Subepicardial haemorrhages; fatty degeneration heart; lungs, red hepatization with commencing grey hepatization, and emphysema; hydrothorax; hydropericard; P.M. changes, fatty degeneration liver; slight glottis oedema; hyperaemia caecum; localized hyperaemia abomasum; catarrhal duodenitis. (Specimen No. 8433.)
20504	24.9.28	26.9.28	Cyanosis respiratory tract; hyperaemia and oedema lungs; froth in windpipe; fatty changes liver; hyperaemia liver and kidneys; ecchymosis epicard; haemonchosis abomasum; parasitic nodules intestines. (Specimen No. 8472.)
20408	24.9.28	26.9.28	Hyperaemia and oedema lungs; emphysema of the mediastinal region; slight fatty changes liver; slight hyperaemia kidneys; few parasitic nodules intestines. (Specimen No. 8474.)
19892	24.9.28	26.9.28	Hydrothorax; hydropericard; dilatation of heart; severe pulmonary hyperaemia; general cyanosis; swelling and degenerative changes liver and kidneys; glottis oedema; gelatinous infiltration of peritracheal tissues.
21128	24.9.28	27.9.28	Post-mortem changes present; marked hydropericard: extensive hyperaemia and oedema lungs; degeneration liver and kidneys.
20997	24.9.28	27.9.28	Hydrothorax; hydropericard; marked hyperaemia and slight oedema lungs; fatty degeneration and slight parasitic cholangitis; slight hyperaemia and degeneration kidney. A slight acute catarrhal laryngitis and pharyngitis. A slight localized acute catarrhal enteritis. Emphysema mediastinum. (Specimen No. 8478.)
20191	24.9.28	27.9.28	Marked hyperaemia and oedema lungs; slight hyperaemia of pharynx and larynx; slight oedema of epiglottic folds; degeneration of liver; slight hyperaemia and degeneration kidneys; malformation small intestines; fairly extensive emphysema mediastinum and especially peritoneum. (Specimen No. 8479.)

No. of Sheep.	Dipped on.	Date of Death.	Pathological Anatomical Diagnosis.
20407	24.9.28	27.9.28	Marked hydropericard and hydrothorax; well-marked emphysema of mediastinum and less marked in peritoneum; extensive degeneration liver; dilatation of right ventricle; slight degeneration kidneys; numerous parasitic nodules intestines. (Specimen No. 8480.)
21000	24.9.28	27.9.28	Hydrothorax; hydropericard; marked hyperaemia and oedema lungs; emphysema mediastinum; extensive degeneration liver; slight degeneration kidneys; few parasitic nodules intestines.
21091	24.9.28	27.9.28	Marred by decomposition; marked hydrothorax, and hydropericard; emphysema of mediastinum; extensive hyperaemia and oedema lungs.
19918	24.9.28	27.9.28	Hydropericard; hydrothorax; dilatation heart; marked oedema and pulmonary hyperaemia; hyperaemia and degeneration liver.
19562	24.9.28	27.9.28	Cyanosis of subcutis; hyperaemia kidneys, swelling and degeneration liver; hydrothorax; hydropericard; emphysema of mediastinum; degeneration myocard; marked oedema and pulmonary congestion; emphysema peritracheal tissues.
19902	24.9.28	28.9.28	Marked hydropericard and hydrothorax; marked hyperaemia and oedema lungs; degeneration kidneys and liver; slight tumor splenis; slight acute catarrhal enteritis; petechiae epicard.
20963	24.9.28	28.9.28	Hydropericard; emphysema visceral pleura; hyperaemia pharynx and larynx; well-marked hyperaemia and oedema lungs; hyperaemia and degeneration liver and kidneys. (Specimen No. 8497.)
19893	24.9.28	29.9.28	Petechiae epicard; very marked hyperaemia and oedema lungs; petechiae bronchi; fair changes liver, spleen, and kidneys; cysticercus tenuicollis cyst bladder; oesophagostomum nodules; emphysema lungs; gas under pleura.
19903	24.9.28	29.9.28	Hyperaemia, emphysema, oedema lungs; degeneration, hyperaemia liver; hyperaemia kidneys; sub-pleural emphysema; oesophagostomum nodules. (Specimen No. 8501.)

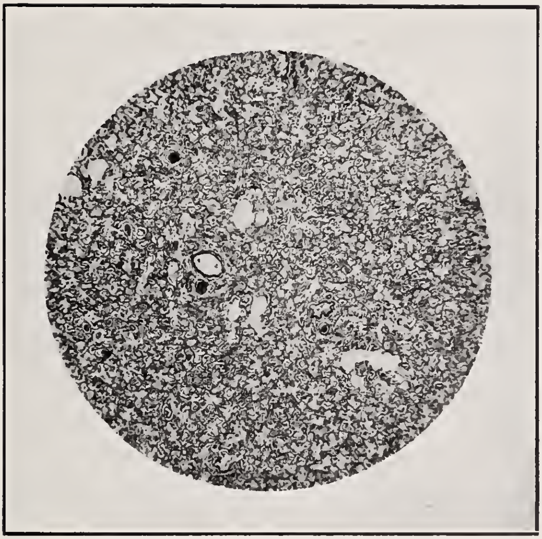

Fig. 1.

Specimen No. 8501. Lung of sheep (magnified 28 ×). Note the cubical transformation of the alveolar epithelium. Some of this epithelium has been shed into the lumen of the alveolus.

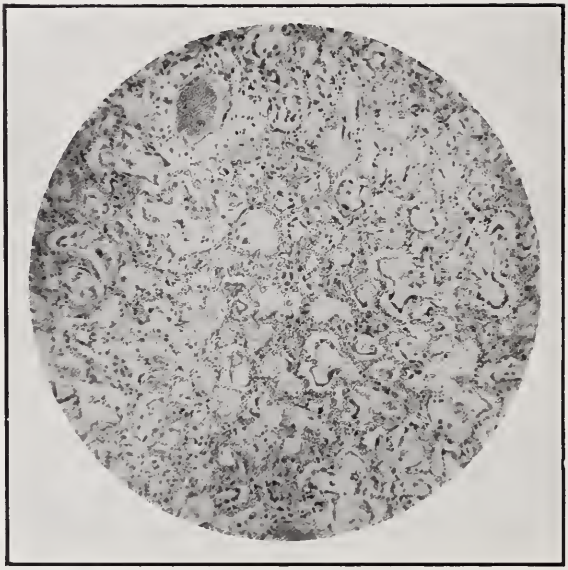

FIG. 2.

Specimen No. 8501. Lung of sheep (magnified 100 ×). Note the cubical transformation of the alveolar epithelium. Some of this epithelium has been shed into the lumen of the alveolus, but is more marked.

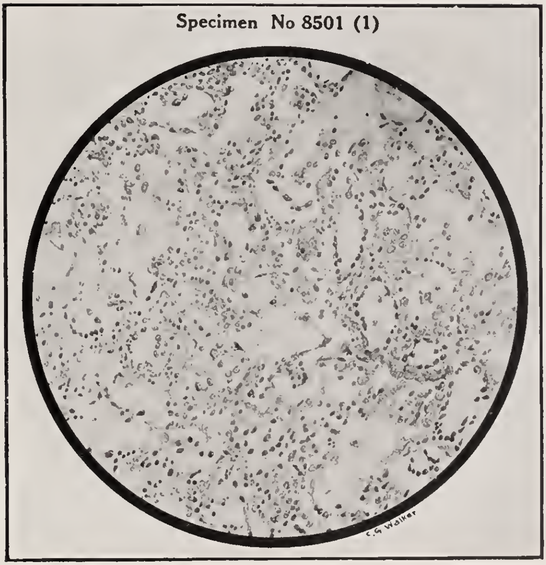

Fig. 3.

Specimen No. 8501. Lung of sheep (magnified 180 ×). Note the cubical transformation of the alveolar epithelium. Some of this epithelium has been shed into the lumen of the alveolus.

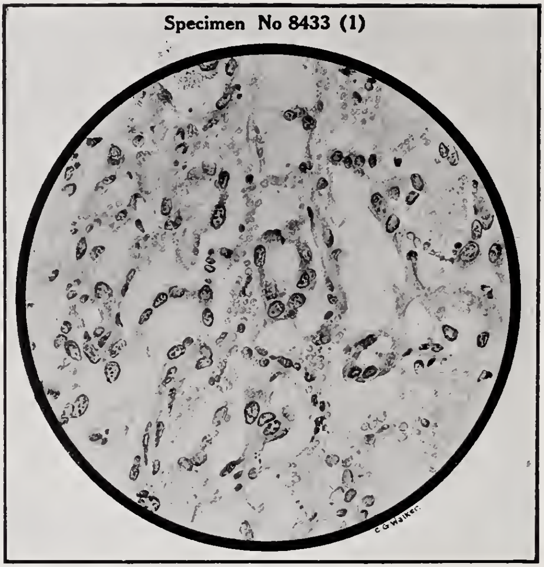

FIG. 4.

Specimen No. 8433. Lung of sheep (magnified 400 ×). Note the cubical transformation of alveolar epithelium, the presence of large amount of blood in the capillaries and boold-vessels, and the air spaces between epithelium and blood-vessels.

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A Note on the Symptomatology of Phenol Poisoning in Sheep induced by certain Dips.

By D. G. STEYN, B.Sc. Dr.Med.Vet., Veterinary Research Officer, Onderstepoort.

As a result of fatalities amongst sheep with certain proprietary phenol dips employed on Mr. Struben's farm, Glen Atholl, Volksrust, and at the Veterinary Research Laboratories at Onderstepoort, Pretoria, it is of interest to note a few points in that respect.

In order to ascertain whether the dipping-fluid caused the deaths in the sheep, Mr. Struben carried out an experimental dipping with fifty-two 3-4 months' old lambs, twenty-six of which were dipped in the suspected dipping-fluid and twenty-six in pure water, in order to exclude climatic effects. Of each of these two groups of sheep thirteen were placed in a shed and thirteen allowed to graze after dipping. This test was carried out in order to ascertain whether the kraaling of the sheep after dipping had any deleterious effect on the animals, and also to exclude the possibility of any intercurrent disease.

The result of this dipping was that the twenty-six sheep dipped in pure water remained healthy, whereas heavy mortality occurred amongst both groups of sheep dipped in the dipping fluid. Placing the sheep in a shed after dipping consequently plays no appreciable part in causing death. Needless to say, the dipping-fluid was prepared according to the instructions on the drums.

In the case of the dip used by Mr. Struben, there was a warning on the drum-label that hard water should not be used in mixing the dip, as it prevents the dip from mixing well with the water, and may consequently give rise to poisoning. A sample of the water was tested for hardness according to the instructions on the drum-label and was found to be negative.

Chemically phenol was detected in the stomach and intestinal walls, the lungs and liver.

Also at Onderstepoort heavy mortality occurred amongst sheep dipped in a proprietary phenol dip, and this formed the basis of a number of dipping experiments which were carried out under the personal supervision of Dr. De Kock. In these experiments the heads of one group of sheep were immersed, whereas in the other group the heads were kept above the water to exclude every possibility of the animals swallowing the dipping-fluid. This was undertaken to ascertain whether the poisoning had occurred by aborption of the dipping-fluid through the skin.

There was a heavy mortality (± 60 per cent.) in both groups of sheep. These experiments rendered definite proof that the animals were poisoned by absorption of the dipping-fluid through the skin. Samples of the dip were tested before and after dipping, and care was taken to prepare the dip according to the instructions issued by the company marketing this product.

Symptoms.—These start within ten to forty-eight hours after the dipping had taken place. The first symptom noticeable is the laboured respiration, which varies from sixty to eighty-eight per minute, and is costo-abdominal. The affected animals are very apathetic, unwilling to move, and there is a bilateral dirty brown muco-purulent discharge from the nose, as well as marked salivation. The breath smells strongly of carbolic acid, and the visible mucous membranes are cyanotic. The pulse is comparatively strong in the beginning, varying from a hundred to hundred and forty-eight per minute. Later on it becomes weak, extremely accelerated and even imperceptible. The temperature varies from 100.2° F. to 103.8° F. The animals stand with an arched back and half-closed eyes. The dyspnoea at this stage is very severe, and the sheep make peculiar grunting noises which can be heard some distance away. On auscultation and percussion of the lungs a pronounced oedema is found to be present. The above symptoms increase in severity until the animals lie prostrate, unable to rise or to move a limb, being in a comatosed state with a subnormal temperature. In this condition the pulse is imperceptible. A few animals showed clonic spasms of different parts of the body. The animals die in a state of complete paralysis and unconsciousness fifteen hours to a few days after the symptoms had set in.

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Skin Cancer of the Angora Goat in South Africa.

By A. D. THOMAS, B.V.Sc., Research Officer, Veterinary Research Laboratory, Onderstepoort, South Africa.

[*Thesis approved for the degree of D.V.Sc. by the University of South Africa.*]

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15th Annual Report of the Director of Veterinary Services, Union of South Africa. October, 1929.

Skin Cancer of the Angora Goat in South Africa.

By A. D. THOMAS, B.V.Sc., Research Officer, Veterinary Research Laboratory, Onderstepoort, South Africa.

I. INTRODUCTION.

CANCER as a disease of the lower animals has in the past scarcely received the attention that it deserves. It has been customary to assume that its incidence was negligible as compared to that in man. From evidence accumulated in the last few years, and from the findings presented in this paper, it will be seen that this assumption no longer seems justifiable.

We know, for instance, that neoplasms of the most varied nature occur in the domestic fowl with relative frequency. Apart from artificially produced tumours, many occur in the smaller laboratory animals, notably rats and mice. With regard to the incidence of neoplasms in the larger domestic mammals, on the other hand, and more especially the ruminants, very little is known. It is true, to a certain extent, that these animals are usually killed for human consumption, or for economic reasons, long before they reach the age at which tumours usually develop, but one cannot dispute the fact that many do reach a comparatively old age. These are usually animals, e.g. milch cows, wool sheep, mohair goats, etc., which during life yield valuable products. Although tumours have from time to time been recorded from these animals by various workers, the information given is often very scanty and incomplete. The histological description is either very brief or lacking altogether, so that even the diagnosis is frequently left in doubt. It is largely on accounts of this paucity of actual detailed histological work in the veterinary literature that one very often experiences real difficulties when confronted with any but the commoner and better-known types of neoplasms. One is thus forced to fall back on to the fairly comprehensive treatises on human neoplastic disease, and draw comparisons and conclusions as best as one can.

In South Africa experience indicates that tumours in the lower animals are by no means a rarity. In fact, during the course of ordinary routine histological examination in the pathological section of this Institute, no less than 123 were returned as neoplasms in the last two years, out of a total of 1,950 specimens examined. It may be of interest to indicate very briefly how these affected the various species of animals.

	Carcinoma.	*Sarcoma.*	*Others.*	*Total.*
Bovine	14	5	15	34
Equine	4	4	14	22
Sheep	4	4	—	8
Goat	16	—	—	16
Dog	8	1	1	10
Pig	—	—	3	3
Monkey	1	—	—	1
Fowl	—	—	29	29
TOTALS	47	14	62	123

Numbers of melanomata from grey horses encountered at post-mortem during this period are not included in the above figures, as it is not customary to collect them for microscopical examination.

The 16 goat neoplasms are those forming the subject of this paper. The collection of fowl tumours has only recently been started, so that the figure above is hardly representative. Since the material has only been partly studied, they are all for the present grouped together under the heading " others."

It is probable that, with the co-operation of the various slaughter-houses and the field veterinarians of this country, much more material could be collected. A number of these tumours were found to be extremely interesting, and are to the best of our knowledge undescribed, thus offering unlimited scope for research work. Indeed, one cannot help being struck by the wealth of material obtainable at the cost of a little trouble and patience—material which, if subjected to systematic study, would undoubtedly open up possibilities of throwing more light on one of the most baffling problems of human and animal pathology.

The present paper is concerned only with certain skin tumours observed in Angora goats under South African conditions.

In 1926, our attention was drawn to the annual losses caused amongst Angora goats in this country by the fairly frequent occurrence of skin tumours. At the very outset it became evident that this breed was particularly susceptible to certain forms of malignant skin tumours. These for the greater part affect the perineum of the females, but also to a much lesser extent the ears, base of horns, and other portions of the epidermis. The condition presents many interesting features, and, if we exclude the well-known adenocarcinoma of the anal glands in the dog, has probably no parallel amongst our domestic animals. As far as could be ascertained, the condition has not yet been recorded. In the first place, the frequency of these tumours in itself forms an interesting observation in view of the belief that animals generally, and ruminants in particular, are only slightly susceptible to malignant growths. The well-known fact that dogs are subject to cancer to a greater degree than other animals is generally attributed to the greater age these are allowed to attain as pets. This, however, is open to objection, as a great many other animals are often allowed to reach a ripe old age, either for sentimental or other reasons, and yet probably show much less tendency towards tumour formation than dogs. Age is obviously not the only predisposing factor. Breed is just as important, and the susceptibility of different species and even varieties of the same species varies greatly. One need only mention, for example, the well-known melanomata in grey horses. The frequency of these tumours under conditions of age, locality, food, work, etc., quite similar for grey as well as for other horses, shows clearly that the susceptibility, whatever it may be, is inherent in the grey horse itself. The Angora goat, as will become evident from the observations to follow, presents a remotely similar example. Apart from the fact that these tumours are often melanotic and appear frequently in the region of perineum in both horses and goats, it will be seen that they have nothing in common. In the horse they are melano-sarcomata, whereas those in the goat are of epithelial origin. In comparing the native and Boer goats of this country with the Angora, under similar conditions of environments and age, often even mixed in the same flock, we find that the former, with very few exceptions, never suffer from cancer, whereas in the latter its frequency is relatively high. This is so striking that many farmers consider the condition contagious, and deal with it accordingly. One must say, however, that, except for an occasional valuable stud animal, the losses due to cancer are not considered very great when compared with the occasional heavy annual mortality resulting from droughts, poisonous plants, and other agencies common to this country. This probably explains why the condition has not attracted the attention of veterinarians sooner. Mention of the condition from one particularly observant farmer in correspondence with this Institute led to the discovery that it was fairly general in Angora flocks, in which a very small percentage of cases occur regularly every year.

It was decided to investigate this interesting condition, and the results which have been obtained to date are given in this paper. The main object of this article is the description of the tumours collected. Incidental notes on the clinical aspects and treatment, gathered partly from the very limited field experience, are added where possible. A few observations and suggestions regarding possible predisposing and causative factors are also given. The experimental part of the work is naturally restricted, especially when dealing with large animals as in this case. Such experiments necessarily extend over very long periods of time, a fact which further curtails the scope and variety of experiments which can be undertaken.

From inquiries made amongst Angora farmers themselves, this disease must have existed in the Cape ever since the early days of the Angora goat in this country. The term " cancer," " canker," or its Afrikaans equivalent " kanker," is generally used to designate any tumours with malignant tendencies. Amongst Angora goat farmers, however, it has become a specific term for a malignant disease which usually affects the perineum, sometimes the ears or other parts of skin of these animals. Frequent metastases, and symptoms of cachexia which accompany these tumours in their ultimate fatal form, certainly justify the term cancer, and also the belief that the disease is incurable in its advanced stage. A great deal of information was collected from many farmers who had had to deal with the condition. Most of it, however, although often interesting, was found to be very conflicting and unreliable, and had to be discarded.

II. THE SOUTH AFRICAN ANGORA GOAT.

ORIGIN.

In order to appreciate certain points raised later in this paper, it is perhaps relevant at this stage to recapitulate briefly the history of the introduction of this animal into South Africa. This incidentally led to the establishing of one of the most remunerative lines of stock-farming in the more or less arid regions of the Cape. Cronwright Schreiner states that the first importation of one Angora she-goat with 12 males from Asia took place in 1838. These rams eventually all proved sterile, having evidently been tampered with before their departure from their home country.

Fortunately, the she-goat, during the course of the voyage, gave birth to a ram kid, so that this ram with its mother really formed the nucleus of the South African Angora goat industry. Many more and larger consignments were subsequently imported, i.e. in 1856, 1857, 1858, and later. Practically all these earlier imported goats were at first freely crossed with the native and Boer goat, which are known for their hardiness and prolificacy (twins and even triplets being quite common). This cross, although it undoubtedly meant a serious setback in the quality and quantity of mohair, has had a marked beneficial effect in producing a hardier race, aud rapid multiplication at the beginning. Subsequent grading up by selection resulted in the present-day relatively high standard animal. One, however, often notices reversions to coarse hair, pigmented skin, hair, etc., as a result of the impure original stock.

HABITAT.

The Angora goat population of the Union at present numbers some 1,825,000 animals. Although new blood was several times introduced from Turkey, inbreeding necessarily had to be resorted to in this grading up process, in order to bring the quality of the mohair to that high standard required for the trade. It is probably on account of this high specialization for the production of mohair of exceptional quality that the Angora goat, like other highly bred animals, presents such refined characters and is rather delicate in constitution. Angora goats do fairly well in many parts of the Union, but for the production of the best mohair, certain very definite climatic and pastoral conditions are essential, and these are to be found in the colder, dry, semi-arid districts of the Cape. In these regions infectious diseases are practically unknown, but the periodic severe droughts and attendant ills, such as over-stocking and plant poisoning, cause at times considerable losses. The goats are usually herded in large flocks by day, and kraaled at night.

This practice is being gradually replaced by a more modern and rational system of allowing animals free range day and night in vermin-proof camps. As a matter of fact, the Angora goat is no worse off as regards hygienic and other conditions than sheep and native goats kept in the same locality.

NORMAL SKIN.

The better-bred Angora goat has as a rule a clear, pinkish white skin of fine pliable texture. The long, uniform, wavy, silky locks of pure white mohair cover the whole body with the exception of the face, ears, and distal extremities of the limbs. The latter parts are covered by coarse, short, stubby hair. Very often, however, blackish pigmented patches are present on the skin, especially that of face, ears, and perineum, rarely on the body. These vary in size from that of a pin's head to half a crown or larger, and in number from a few scattered patches to great numbers often tending to confluence. The hair over these is as a rule white. Occasionally one sees goats with completely pigmented skin; the hairy coat then has a bluish grey colour. This distribution of pigment in the skin is important as it probably has a bearing on the origin of the pigmented tumours to be mentioned later.

The skin is usually dry and often covered with fine epithelial scabs resembling dandruff. Oily skins are sometimes met with, but such animals are culled, as their mohair is considered coarse. The skin of the face and ears is rather thicker, and very often pigmented: in fact, the skin around the eyes is nearly always so. The ears often show laceration and snicks, the result of ear-marking. The perineal skin is soft and of a pinkish grey to very light brown colour. It is well supplied with sweat and sebaceous glands, but only sparsely covered with fine hair. The recess below the tail usually contains a variable quantity of greyish, cheesy, sour-smelling matter resembling smegma. This may be in the form of a soft, pultaceous mass, it may be caked or even dry and crumbly, but always fairly firmly adherent to the underlying skin. Little balls of this substance matted together with dirt and formed by the to and fro rubbing of the tail, can often be seen hanging from small excrescences of the epidermis in this region. The skin in this recess, once cleaned, is very thin and hairless, white in colour, and pitted by the openings of countless sebaceous glands.

HISTOLOGY OF THE SKIN.

For the purpose of obtaining an idea of the histology of the skin of those parts especially subject to cancer, an apparently healthy, mature she-goat (14433) was killed, and material for examination collected from the non-pigmented perineal skin. Sections were prepared from 13 different pieces, representing regularly spaced layers on either side of a median line from the depth of the tail recess down to the region of the clitoris. The skin just under the tail, i.e. in the depth of the recess, shows numerous very large sebaceous glands, nearly touching each other. They show branched sacculations, which communicate with the surface by wide funnel-shaped opening in the epidermis. Hair follicles are practically absent in this region, but the sweat glands are well developed. The epidermis is fairly thin and covered by very little keratinized substance. In the sections taken at the level of the anus, the epidermal layer becomes fairly thick, and in many places, especially along some of the follicles, evidence of hornifying centres can be seen. Still further down the hair follicles becomes numerous. Sebaceous and sweat glands remain well developed and numerous, although not quite so close together, nor so large as above. In the region between the anus and the vulva, situated well below the level of the sweat glands, and lying on the bundles of muscle fibres, are sparse but well-developed glands, which appear to have a special function. Their lumen contains an eosinophilic, homogeneous material resembling colloid. In appearance they resemble the sweat glands to some extent, but show many more convolutions. Some of the tubules often show branching sacculations. The lining epithelium consists of a single layer of low cubical epithelium often having a peculiar, vacuolate appearance due to the presence of many refractory globules in the cytoplasm. The opening of these glands was not traced. They are probably modified skin glands, of the nature of the so-called apocrine sweat gland. Pigment is absent, or very scarce, in the basal layer of the epidermis, but may be seen in chromatophores lying just under the basal membrane. These cells are few in number, and fairly well spaced out, being more frequent between the sebaceous glands and around the hair follicles. Throughout this region, round cells in clusters, or in diffuse arrangement, are seen in the corium, sometimes associated with the interpapillary processes of the epidermis. These round cell accumulations are more evident under the squamous epithelium lining the vulva. Here they seem actually to invade the epithelium in places, and are often mixed with neutrophiles. The chromatophores are generally absent from this region. Towards the periphery of the perineum, there is a gradual decrease in the number and size of the skin glands as the hair follicles become more numerous.

Specimens of skin from other goats, some with pigmented perineum and ears, were also examined. The histological picture is similar to the above in all essentials, except that the distribution of pigment is different. The pigmented patches or maculae mentioned above contain a good deal of melanin. The granules are most abundant in the germinal layer, but pervade also the whole malpighian layer.

The other parts of skin may contain smaller but variable amounts of melanin, which is either uniformly spread along the basal layer, or confined to certain areas only. The arrangement of the skin glands is the same. They are large and closer together, often unassociated with hair follicles near the anus and under the tail, whereas they gradually become smaller towards the periphery of the perineum. Here and there, near the anus and vulva, minute superficial erosions with infiltration of neutrophiles and round cells under and into the epidermis are seen. Sometimes a layer of inflammatory exudate and fibrin covers the excoriation. The skin of the ear from two specimens examined presents nothing unusual. The epidermis is thrown into coarse serrations superficially, except on the inner surface of ear, where it is relatively smooth. The interpapillary projections are small and shallow, but at the edge of the ear may reach considerable depth and thickness (acanthosis). The basal layer of the epidermis and hair follicles, often also the stratum spinosum, shows a more or less heavy pigmentation, often patchy in distribution. The inner surface of the ear generally shows much less pigmentation. The sebaceous glands are fairly frequent, but small, and open into the numerous hair follicles present. Sweat glands are rare and very small.

III. SKIN CANCER OF ANGORA GOATS.

Before proceeding with the description of the cases observed, it is perhaps advisable to enumerate the various circumstances under which these tumours occur, the types of animals affected, and how the farmer deals with them.

OCCURRENCE

Species.—As pointed out, only Angora goats are susceptible to these types of tumour to this extent. Evidence gathered from farmers, as well as personal observations, show that new growths of this description are practically unknown in the South African native or Boer goat.

In sheep, skin tumours are occasionally met with on the head in various situations, such as on the ears, eyelids, cheeks, and forehead, etc. Several such cases have already been examined at this Institute and identified as spino-cellular carcinomata. Dodd (1923) describes the occurrence of skin cancer of the ear in Australian sheep, while Beatti attributes the carcinoma seen in Argentine sheep to injuries from thorn-bushes.

SEX.

She-goats are by far the most commonly affected. Kapaters or castrated males only rarely, and then usually on a broken horn stump, ear, and elsewhere on the skin. Rams, on the other hand, have apparently never been observed suffering from cancer. It should be remembered that rams usually form only a small percentage of the flocks. Whether this has actually anything to do with the apparent total absence of tumours in them cannot at this stage be said, but is certainly seems that males, including kapaters, are much less susceptible to these tumours than females.

AGE.

Accurate figures cannot be given, but most of the cases observed were adult and aged animals. Although some " warts " were seen on quite young goats, these were merely of papillomatous nature.

FREQUENCY.

The estimated percentage occurrence of cancer amongst goats, as stated by various farmers consulted, ranges from 1 per mil. to 5 per cent. of the flock yearly. Unfortunately, no statistics of any description are available to substantiate or refute these figures, which are undoubtedly very rough estimates, and often on the high side. Even from the flocks inspected personally, it was found impossible to arrive at any definite figure, since the exact number of affected animals destroyed by attendants during the season could not be ascertained. It is very difficult to examine all the goats in the large and scattered flocks, to make sure that no early cases are missed. The cases actually studied were nearly all obtained from one owner, who estimates the percentage at 3 per cent. This figure, however, seems unwarrantably high. The correct figure varies from flock to flock, and from year to year within the ranges mentioned above, and probably rarely reaches or exceeds 1 per cent.

SEASON.

Tumours apparently develop at any time of the year, although some farmers maintain that they are more frequent at, or after, tupping time. In fact, they firmly believe that the disease is transmitted by the ram at service, from affected she-goats, to the healthy. Although experiments to prove this point could not actually be carried out, it seems very unlikely that this is the case. (See Contact Experiments.)

LOCALITY.

These tumours apparently occur all over the country where Angora goats are kept, even in the Transvaal under climatic conditions totally different to those of the Cape.

GENERAL APPEARANCE.

Under farming conditions the flocks are brought in at certain periods, e.g. for shearing, at kidding time, for dipping, etc. This affords an opportunity of catching and handling each animal, so that it can be subjected to a more or less close inspection. In this way many of the tumours are detected while still fairly small. Tumours otherwise have to reach a certain size before they can readily be detected without handling the animal. As a rule, the first indication of the presence of a tumour is the soiling and matting together of hair around the perineum with slimy exudate and dirt. Tumours in other parts are also generally detected when they become ulcerated, start giving off soiling, stinking exudates, and become fly-struck. The general appearance of such tumours naturally varies greatly. The size may be anything from that of a pea up to 10 cm. in diameter, either rounded and lobulated, or flattened like a great raised ulcer. The parts affected in order of frequency are the perineum, ear, horn stump, udder, and rest of skin, the first being by far the most commonly affected. Even in this situation the tumours have predilection seats, either the skin of the recess under the tail is affected or the sides of the vulva. Very often both are affected so that the growths run into each other. The tumours under the tail usually originate in, and are attached to, the skin above the anus, either in the centre or on to the side. The area of attachment may remain small, so that the tumour as it grows becomes pedunculate and mushroom-like, or else, on the other hand, the base may widen with the tumour, and on account of necrosis or the action of fly larvae it may assume the form of a wide, shallow, ragged ulcer with indurated, raised edges. From the usual starting point, i.e. the depth of the recess under the tail, the tumour grows as a moist, pinkish mass, sometimes greyish to black, until it fills the whole space between the two lateral tail folds, which may even be pushed outward. It may then extend downwards, hanging over, or on either side of the anus, and even down to the vulva. The vulvar tumours are more usually of the rodent ulcer type, partly covered with crusts, and gradually widening and getting deeper, or bulging out to take on a cauliflower appearance. It seems to be a common property of these tumours that, as soon as they reach a certain size, they tend to become surrounded and covered by moist exudate, which forms a suitable pabulum for bacteria and other organisms. This exudate tends to soil and mat the adjoining hair and contributes to considerable irritation. In some cases it appears to have a macerating influence on the lower lying or in-contact skin, often to such an extent as to give rise to actual newgrowth or contact implantation. Blow-flies are attracted to this region by the foul smell and moistness. They deposit their eggs on the tumour, with the result that the emerging maggots soon attack the tumour and eat their way into its substance, causing intense irritation and suffering to the animal. Apart from fly larvae, the tumours often undergo loss of substance by the sloughing off of gangrenous tissue. Crusts which form over them are torn off mechanically, either accidentally by horning, or by the animal itself while scratching or biting itself on account of irritation. Haemorrhage is also very easily set up by the least traumatic action on the tumour. The early stage of the tumour, i.e. from the time it begins developing until it becomes large enough to cause exudation and irritation, occupies a varying length of time up to several months. During this period the animal seems to experience little or no inconvenience and certainly remains in fair or even good condition, according to the state of the pasture. Such a small tumour, especially when in the depth of the tail recess, can be easily overlooked, unless the tail is lifted and the part closely examined. In most cases the tumour grows only very slowly at first, and may even at times show considerable regression (gangrenous sloughs, maggots, trauma). On reaching a certain size, however, or for other unknown reasons, the disease may suddenly take a more rapid course. The animal stands apart from others, does not feed properly, and shows all the signs of intense worry and irritation, namely, constantly flicking its tail, stamping its feet, biting, horning, and scratching the affected part, sometimes furiously, or else biting viciously other goats around the perineum. The animal seeks dark corners to lie in, or creeps amongst other goats, and usually lies with its head towards its perineum in an attempt to ward off flies. Condition is lost very rapidly, and the animal finally dies in a state of extreme emaciation and cachexia. In practice, fortunately, affected animals are nearly always spared this cruel and slow torture, or at any rate it is curtailed. The farmer realizes from experience that the condition is incurable. All cases are, therefore, destroyed as soon as they come to his notice. In this way he is able to make use of the carcase while still in fair condition, and incidentally saves the animals from unnecessary suffering. She-goats affected with perineal tumours, if allowed to breed, naturally undergo terrible suffering and laceration of vulva during parturition. Apart from these humane and economic reasons, early culling is practised to minimize the chances of carrying over the affection from diseased animals to clean ones.

The economic losses due to cancer of the goat in the past have been overshadowed by those due to other causes. The veterinarians were mostly preoccupied with more important and urgent stock diseases. This probably is the reason why the condition has not been recorded before. Malignant growths in their various forms are well known and accepted as inevitable, or taken for granted, and as they are known to be incurable, no further notice is taken of them beyond destroying the affected animals. Attempts at treatment have been repeatedly tried by the more enterprising owners, especially where the more valuable animals are concerned. A paste made up with Cooper's arsenical dipping powder is said to have some beneficial effect when applied in the early stages. Other drugs and herbs have also been tried with varying and doubtful results. Surgical removal in the crudest form is practised in some rare cases of cancer of the ear, by the simple process of cutting off the ear above the tumour. A few " permanent " cures were effected in this way. Such cases, however, are few and far between, especially in late years, as most farmers prefer to kill off all affected animals. They claim that this is directly responsible for the undoubted reduction in incidence of cancer lately.

There are also some who maintain that since the advent of a more modern and rational system of farm management, i.e. allowing the animals free range in vermin-proof camps, instead of herding and kraaling, coupled, of course, with the practice of culling, the occurrence of cancer has been materially reduced.

MATERIAL COLLECTED.

The material studied consisted of—

(1) seven tumours, mostly in their early stage, removed surgically from affected animals on the owner's farm (Series 7256). This incidentally afforded an opportunity of visiting the flocks under actual farming conditions, and of gathering first-hand information. Other information was obtained from various progressive Angora goat farmers by circulating a questionaire dealing with some of the most obvious aspects of this disease;

(2) fifteen affected animals obtained from various sources, of which 11 were from the owner referred to above.* These animals were consigned to this Institute and kept in stables and later together with some 30 experimental animals in a small paddock. Some were used for transmission and various other experiments, while others were kept for the observation of the tumours in their various stages of development. In nearly all cases material was collected for histological examination. The description of each case, including the clinical observation, post-mortem and histological results, will be found in protocol form at the end of this paper.

The following table gives a summary of the cases:—

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* Since going to press, 21 more cases of cancerous goats have been collected, of which 14 were affected at the perineum and 7 on the ears.

TABLE OF CASES.

Case.	Sex.	Age.	Situation.	Diagnosis.	Metastases.	Pigment.	Maggots.	Remarks.
14771	Female	F.m.	Perineum	Mixed sp. and bas. c. ca.	—	XX	—	Died, probably poisoning.
14772	,,	Aged	Anus	Bas. c. ca.	—	—	—	Tumour removed. R.
14773	,,	,,	Perineum	Mixed bas. and sp. c. ca.	—	XXX	—	Killed.
17293	,,	,,	Perineum	Sp. c. ca.	XX	—	XXX	Died.
17294	,,	,,	Anus	Mixed sp. and bas. c. ca.	—	—	—	Tumour removed surgically. R.
17296	,,	,,	Horn stump	Bas. c. ca.	XXX	XXX	XXX	Killed *in extremis.*
17297	,,	,,	Vulva	Bas. c. ca.	XXX	XX	XXX	Died.
17298	,,	,,	Vulva	Sp. c. ca.	XX	—	XX	Died.
17299	,,	,,	Ear	Bas. c. ca.	—	XXX	XX	Tumour removed surgically. R.
21957	,,	,,	Perineum	Mixed sp. and bas. c. ca.	?	—	?	Biopsy for diagnosis.
7256A	,,	,,	Anus	Bas. c. ca.	—	X	—	Tumour removed surgically. R.
7256B	,,	4 t.	Vulva	Bas. c. ca.	—	XXX	—	Tumour removed surgically. R.
7256C	,,	Aged	Anus	Mixed bas. and sp. c. ca.	—	XX	—	Tumour removed surgically. R.
7256D	,,	F.m.	Anus	Bas. c. ca.	—	X	—	Tumour removed surgically. R.
7256E	,,	6 t.	Neck	Bas. c. ca.	—	—	X	Tumour removed surgically. R.
7256F	,,	Aged	Anus	Sp. c. ca.	—	—	XXX	Tumour removed surgically. R.
14770	,,	F.m.	Anus	Clinically bas. c. ca.	—	—	XXX	Tumour eaten up by maggots. R.
17292	Male	,,	Horn stump	Clinically bas. c. ca.	—	—	XXX	Died. No material collected.
17300	Female	2 t.	Forehead	Papilloma	—	—	—	Spontaneous disappearance. R.
17301	,,	,,	Face	Papilloma	—	—	—	Spontaneous disappearance. R.
9983	,,	Aged	Anus	Mixed sp. and bas. c. ca. (?)	—	—	—	Died.
7256G	,,	6 t.	Vulva	Chron. pur. dermatitis	—	—	—	Died. Early stage tumour?
14505	,,	Aged	Vulva	—	—	—	—	Under observation. See protocol.
15690	,,	,,	Anus	—	—	—	—	Under observation. See protocol.

Bas. c. ca. = Basal cell carcinoma.
Sp. c. ca. = Spinous cell carcinoma.
F.m. = Full mouth.
2 t. = Two tooth.
R. = Recovered.

THE CASES STUDIED.

From the foregoing table it will be seen that 24 animals are included, although they did not all suffer from true cancer. Nos. 14770-14773 were received here on or about the 20th August, 1926, but were not taken over by the writer until the 17th June, 1927. A further 10 goats, Nos. 17292-17301, were received from the same source on the 15th June, 1927. They were selected and sent up by the owner as specimens of cancer cases. No. 17295 is not included in the table, as on arrival no tumour could be found on it, neither did any develop subsequently. Nos. 17300 and 17301 were young 2-tooth animals, and had papillomata, which subsequently disappeared. It is stated that "warts" by being continually scratched and injured may develop into cancer, but it is doubtful whether these would have become true cancers under any circumstances. The "warts" that the farmers may have seen turn to cancer are probably not ordinary papillomata, but cancerous or precancerous growths. No. 17292 died on arrival, but unfortunately no material was collected for section as it was not realized in time that the gangrenous tissue around the broken horn stump might be of the nature of a tumour. It is quite probable that this case was similar to 17296. No. 21957, received on the 17th November, 1928, from a different locality, was suffering from a typical cancer of the perineum. The seven specimens marked 7256 A to 7256 G are all tumours removed on the 20th August, 1927, during the course of a visit to the farm from which the first two lots originated. These tumours had developed, or rather become apparent to casual inspection, since the previous series (17292-17301) had been culled from the flocks. It may be taken, therefore, that they were still in the earlier stages of development, a point which seems supported by their relative small size and microscopical appearance generally. The object of this visit was to study the local conditions, collect material for microscopical examination, and incidentally note the curative effects of surgical removal. Nos. 9983, 14505, and 15690 were picked out from the available experimental goats, on account of peculiarities which might throw some light on the mode of development of the tumours. No. 9983 had a small pea-size protuberance under the tail to start with; this eventually grew to a flat excrescence a couple of cms. in diameter. The histology of this growth seems to explain the origin of this type of neoplasm to some extent. No. 14505, which accidentally received a traumatic laceration of the vulva, was left untreated and kept under observation to see whether the chronic inflammation would lead to the beginning of a new growth. Up to date, no evidence of this can be seen. No. 15690 had a peculiar fringe of small, short, skin outgrowths under the tail, on which caked balls of secretion, dirt, etc., accumulated and hung down. These outgrowths have so far made no progress and are apparently malformations of the skin, and not at this stage to be regarded as tumours (see Fig. 45). Out of the 24 animals included in the table, Nos. 17300, 17301, 14505, and 15690 are only of interest inasmuch as the lesions noticed might form a starting point for cancerous growths. Nos. 14770, 17292, 7256 G, and 9983 cannot be taken into account, as no material was examined microscopically from the first two, and the diagnosis of the last two is doubtful. No. 9983 and also 14772 show very interesting changes, which it is believed might explain the earlier developmental stages of the tumours in question.

This leaves a total of 16 goats in which epitheliomata of various types were examined histologically.

The diagnoses arrived at are given in the table, and the clinical and microscopical findings in the protocols at the end of this paper.

These sixteen cases are all females. The only castrated male (Kapater 17292) was excluded on account of lack of histological evidence as to the nature of the growth. Their ages vary; the youngest being 4 tooth, while the majority are classed as full-mouth and aged. The latter term includes those in which the incisors are worn down to mere rounded stumps on the gums.

The tumours were distributed as follows: 13 at perineum, one on a broken horn stump, one on the ear, and one on the neck. By perineum is meant the short-haired skin around anus and vulva. These two organs are not considered separately, since in our experience both may be affected at once. Tumours have been seen extending from the one to the other during the course of their development, e.g. 14771 and 17293.

As regards the nature of the various neoplasms, three had more or less definite cancroid tendencies, and can be called spino-cellular carcinomata. Eight showed no keratinization, and are therefore classed as basal-cell carcinomata, while the remaining five are combined forms, either conaining well-demarcated areas of either type, or else a more or less ill-defined intermediate structure. These still have some of the characters of a basal-cell carcinoma, but some cells show degeneration, swelling, and keratinization, resembling somewhat those seen in cancroids. It must be stated, however, that the above grouping, which is arbitrarily based on one or two characteristics, namely, keratinization and morphology of cells, is very unsatisfactory, since there is no sharp distinction from one to the other, but rather as it were a graded scale of transition from the round-celled, sarcoma-like, basal cell tumour on the one hand, to the pearl nest-forming cancroid on the other.

Krompecher in his classification of Basaliomes of course does make provision for keratinizing basal-cell carcinoma, which he calls " Carcinoma basocellulare parakeratodes."

Metastases were only noted in four of the cases. Two of these, 17293 and 17298, were in the supramammary lymphatic glands only, from primary perineal cancroids. The remaining two cases were from non-keratinizing epitheliomata, i.e. basal-cell carcinoma. No. 17296, with the growth on the broken left horn stump, showed metastases in all the lymphatic glands of the head and neck, on the left side only, as far down as the prescapular, and also the liver (see Fig. 18). The other, 17297, also had extensive metastases. The lymphatic glands affected were the supramammary on both sides, sublumbar and mediastinal, and in addition the kidneys, pancreas, liver, heart, and lungs (see Fig. 22). It is evident thus that malignancy is independent of the hornifying properties of these neoplasms. We have, for instance, several other cases of mixed forms, some of which were observed for a considerable period of time, where metastases did not occur. Pigmentation (melanin) appears to play an important rôle in these tumours. It will be seen that a few had a pitch-black colour, due to presence of large amounts of melanin, which was formed in the growth itself. Some had varying quantities of this pigment, which was sometimes localized in certain parts only, while others again seem to be devoid of it altogether.

BACTERIOLOGY.

The bacteriological aspect of these tumours was investigated only very superficially. On account of the lack of facilities and time, it was felt that this subject was better left over for the time being. There are certainly many interesting points which are well worth investigating by a bacteriologist or protozoologist; for instance, the rôle played by bacteria and spiral organisms in the development or extension of the tumours. The smear preparations made from the scrapings of the surface of the tumours showed an abundant and varied bacterial flora. This could, however, be expected from the open nature of the lesions. In addition, also, many cases showed the presence of numerous spirochaetes and spirilla. These were at first regarded with suspicion, in view of the alleged transmissibility of the condition at tupping time. Attempts at transmission, e.g. by close contact of affected animals with non-affected ones, rubbing of infected material into the mucous membrane of the vulva, scarification, and injection with suitable material, all proved unsuccessful. These spirochaetes are usually to be found in the moist exudate around the tumours. They appear to be always associated with a type of bacterium seen in fusiform or beaded-chain formation. They correspond very closely to those described by Tunnicliff and others, and found in Vincent's angina, otitis media, and adenoids in man. It is, of course, well known too that types of spirochaetes are often found in ulcerating open sores in man and animals generally. In pigs, in this country, they are often found in large skin abscesses, of which they are believed to be the cause. Peculiarly enough, the spirochaetes found on these tumours occurred apparently only in the goats which had been on the premises of this Institute for some time. None could be found on the newly introduced cases, nor on those examined on the farm of origin (7256 series). Furthermore, in those cases where material from swabs of the tumours was injected subcutaneouly near the anus, an abscess usually developed and burst, leaving an open wound in which spirochaetes were often found in large numbers. In spite of this, such a wound invariably healed up rapidly.

From the foregoing it is concluded, therefore, that the spirochaetes observed were only part of the many contaminating saprophytes growing under the favourable conditions produced around the ulcerating tumours, and that they were not directly the cause of the tumours. This, of course, does not preclude the possibility of secondary influence on the further development or spreading of the growth once started, for instance, by the macerating or irritating effects of such organisms on the adjacent tissues.

TREATMENT.

This deals only shortly with the surgical aspect, since the material available was too restricted to try the effects of drugs and other modes of therapy. Surgical removal of the tumour was performed in the following cases:—14772, 7256 A, B, C, D, E, F, and G, 17294, and 17299. It proved entirely successful in so far as can be ascertained at this stage. The operation itself is very simple on account of the superficial attachment and usually non-infiltrative nature of these tumours. Most of the above cases were done, under field conditions, with a local anaesthetic. An injection of 1/10 to ½ grain of cocaine with adrenalin, into the subcutaneous tissue near or under the growth, was found to answer the purpose very well. The tumour and neighbouring skin was, in the first place, washed with soap, warm water, and disinfectant, dried and painted over with tincture of iodine. The tumour could then very easily be dissected out, especially when of the stalked variety, by following and enucleating the firm connective tissue stroma from the subcutis. In one case of the ulcer type, which penetrated deeply into the vulva lip and involved the mucosa (7256 B), it was necessary to cut fairly deeply, in order to remove the whole pigmented tissue. The mucosa was then sutured to the skin. In all these cases the tumours were either near the anus or vulva. The tumour removed from 17299 was situated on the ear. Here, of course, the ear itself was amputated just proximal to the tumour and a few sutures placed at the edge to approximate the skin over the cartilage. In another case (7256 E) the tumour was attached over quite a large area of skin over the neck, resulting after the operation in a fairly wide, though shallow, wound. In all these cases after attending to haemostasis, bleeding being fairly profuse in some, e.g. 7256 B, a few interrupted sutures were placed in position to bring the skin edges together. After-treatment consisted in cleaning and dusting with iodoform a few times. The 7256 series running in the veld were never again attended to. The whole group made a rapid and uneventful recovery. As a matter of interest, the case of goat 14770 may be mentioned here, as it practically is equivalent to surgical removal. Here the maggots which swarmed in the tumour ate their way right down into the base of the very slender stalk, in such a way that the tumour and its occupants fell off. The small wound on the stalk stump healed up soon afterwards and there has been no recurrence. The seven cases done in the field, i.e. 7256 series, were not seen again by the writer, but the owner stated in his last communication that all had recovered from the operation, and were to all outward appearances healthy. Two of these, however, subsequently died from causes unassociated with cancer. Of those which were kept under observation, No. 14772 was killed 25 months after operation on account of poverty and old age. It had no outwardly visible recurrences. Subsequent microscopic examination of the perineal skin shows small foci of epithelial growth which seem to be the beginnings of tumours. The skin also showed distinct atrophy and degeneration. Nos. 14770, 17299, and 17294 are still alive and well, 19, 14, and 2 months respectively after removal of the tumours.

From these few cases it would seem that these skin tumours in Angora goats respond fairly well to surgical treatment. Experience in this respect is, however, limited, and more work in this line has to be done. Close observation of the treated cases under various conditions is necessary before definite conclusions can be drawn. It is, for instance, not known what effect breeding would have on such animals. Mention is made in the literature (Gans 1928) that the basal-cell carcinoma, and even the squamous-cell carcinoma in man, do not readily recur once removed.

In most cases of skin cancer in the goat, therefore, surgical treatment is indicated, provided metastasis has not obviously taken place. The chances of success in the earlier stages are naturally greater, and the operation is quite simple on account of the superficial nature of the growths. Where the presence of metastases is suspected, it may be advisable (on purely theoretical grounds) to remove the supramammary lymphatic glands as well, i.e. if the tumour is at the perineum. These glands, it appears, become involved in metastasis before any other invasion takes place, e.g. (17293 and 17298).

Under the present circumstances of Angora-goat farming in this country, it is doubtful whether curative measures, unless such could be performed by the farmer himself, would be of any practical value. Except in rare cases, the individual value of goats is not great enough and the services of a veterinarian too difficult to obtain to render surgical treatment a practical solution.

The best course to follow at this stage is to encourage the present policy of culling all affected animals as early as possible. Most farmers do this already, and attribute to it the decrease in incidence of cancer in recent years. There would not be any great difficulty in generalizing this practice. Should the belief that the disease is transmissible, or even that it is due to inherited predisposition, be founded, then such a course would, for obvious reasons, be the proper one to adopt. Finally, such a procedure also commends itself from purely hygienic and humanitarian points of view.

As nothing is known regarding the etiology of the condition, little can be said about prevention. It is probable, however, that timely surgical treatment of fractured horns and other injuries might avert some, at least, of the tumours which develop in those situations.

IV. DISCUSSION.

LITERATURE.

The literature relating to skin tumours in the domestic animal covers a surprisingly small field. Fairly comprehensive histological, as well as macroscopical, descriptions of a few types of skin and skin gland neoplasms are given by Joest and by Kitt in their standard works on "Animal Pathology." Apart from these, occasional outstanding cases have been recorded by various writers, but little has apparently been done in attempting to study them thoroughly.

Kitt states that, in the dog, adenomata of the sebaceous glands, and especially of the circumanal glands, are fairly frequent. The skin glands of the ear are also sometimes affected. The size of these adenomata reaches that of a hazel-nut or even that of a small apple, and the skin over them is rarely ulcerated. The circumanal adenoma shows no lumen in its structure, no secretion is present, and there is no outlet. Skin carcinomata, he states, are to be noted in dogs, cats, and horses to a greater extent than in other animals. The predilection seat of development is at, or near, the junction of the epidermis and mucous membranes, e.g. lips in the horse, dog, and cat, anus in the dog. (In this connection it is interesting to note that, in this country, cancroids of the nictitating membrane in horses are sometimes met with. Carcinoma of the eyelid in bovines also occurs with relative frequency.)

Fröhner describes an "Ulcus carcinomatodes circumanale" in the dog as a lumpy, slow-growing tumour, soft to firm in consistence. Its ulcerated surface is covered with slimy, stinking secretion, which macerates the neighbouring skin and sets up inflammation. The base of the tumour is continuous with the skin. Anal carcinomata of this type, he states, are as a rule benign.

According to Joest, skin carcinomata of domestic animals are not rare. In the horse they are mostly to be found at the prepuce, tail, and vulva (the latter from the clitoris). In cats at the lips. In cattle Joest and Biederman describe two cases of vulva carcinoma. Paine, Trotter, and many others also mention carcinoma of the vulva in cattle, usually of the typical cancroid type. Joest also describes a "glandular" or basal cell type of carcinoma which may originate from the skin glands or their ducts. These, he states, are very rare in animals. Histologically they consist of solid, winding, branching, garland-like strands of epithelium. The arrangement of these is very irregular; they are devoid of a lumen, so that they cannot easily be mistaken for an adenoma. The epithelium is composed of many layers and is closely packed in sparse connective tissue. On account of the similarity of these cells to those of the basal layer of the epidermis, Krompecher called them basal-cell carcinomata or Basaliomes. He states that one rarely finds in sections evidence of continuity between the epidermis and tumour cells. Ricker and Schwalbe, quoted by Joest, derive the basal-cell carcinoma from the epithelium of the skin glands.

Joest further gives a short account of adenomata of sebaceous and sweat glands in dogs. The former is characterized by the presence of much-branched sebaceous gland lobes. The centre of each sac consists of the usual fat-laden cells. The periphery is lined by layers of darker cubical epithelial cells, which are difficult to distinguish from epidermal or follicular basal cells. These tumours apparently may affect any part of the skin. The sweat gland adenoma described has such a typically glandular structure that it need not be taken into account here for the purpose of comparison.

In Australia, according to Dodd (1923), sheep often suffer from cancer of the ear. This, it is believed, is due to long-standing chronic irritation, the results of ear-marking.

Beatti likewise believes that skin cancer in Argentinian sheep is caused by the repeated injury from the thorny bushes amongst which the sheep have to graze in the Pampas. The cancers alluded to by the last two workers are presumably cancroids. In South Africa such tumours have also been observed at this Institute. One case was a large squamous-cell carcinoma of the forehead, the other of the ear. Lubarsch and Ostertag give more or less similar statistics regarding skin tumours in animals.

With regard to melanotic epithelial tumours in domestic animals, the following have been recorded: Caylor and Schlotthauer describe melano-epithelioma in three young swine. Melanoma were also observed in pigs by Schöpperl; in cattle by Adams and by Schindelka; and in dogs by Schindelka. Hewlett in India mentions newgrowths on the base of the horns of cattle. These go under the name of " horn-core disease " and in Stockman's opinion are of the nature of epitheliomata. Remarkably enough, the goat is not mentioned in connection with skin tumours.

It is fully realized that the above does not include all that has been written on skin cancer in animals. It merely represents the literature which the writer was in a position to consult in the local library. The most interesting and useful information was obtained from some of the standard textbooks on human tumours and skin diseases.

Borst points out that, in one and the same carcinoma of the skin, one must be prepared to see various stages of epithelium differentiation. This peculiarity is often shown by basal-cell carcinomata. These non-keratinizing epithelial tumours are characterized by more or less undifferentiated epithelial cells having polygonal, round, elongated, or spindle shapes. He considers the " rodent ulcer " of man a special type of this carcinoma, slow growing, more restricted to the superficial tissues, and in which calcification can often be seen. He states also that he saw a pigmented, flat cell carcinoma in the form of a pigmented naevus. This consisted of polygonal cells often arranged in strings or strands, but without showing any tendency to keratinization. He could not establish any connection with the epidermis, and therefore did not feel justified in terming it a melano-carcinoma. An alternative was to call it an endothelioma.

A point of considerable interest is raised by McCallum in connection with basal-cell carcinoma. He states that the malignant epitheliomata may present a different histological alteration of any or all the cells of the epidermis. Their malignancy, therefore, need not merely be the effect of their being derived from a somewhat more differentiated layer of the same cells. Krompecher regards these tumours as growths derived from the lowermost, or basal, layer of the epidermis, for which reason they show no tendency to keratinization or horn-pearl formation. Indeed, he is willing to ascribe certain tumours to the cylindrical or germinal layer, others to the cuboidal or rete layers, and the highly malignant epitheliomata to the more superficial or spiny layer.

Oertel gives a brief but very concise description of Krompecher's skin cancer. These little differentiated epidermal cell tumours, according to him, may also grow in infiltrating fashions. They maintain a much more delicate arborescent manner of growth in the form of thin, delicate cell branches and cell columns, with club shapes or pointed extensions. The cells themselves are also finer, more delicate in appearance, often elongated, even fibrillar or spindle shaped (sarcomatous) so that some regard these tumours as endotheliomata. These growths generally arise in the cutis, *not* in the epidermis, i.e. from the skin appendages such as the hair follicles, etc. They then grow upwards, under, or into the epidermis, which then becomes eroded and collapses over the advancing tumour. These cancers ulcerate relatively early, and possess hard, indurated surfaces. Cancroid pearls or any tendency to keratinization of cells are absent, but occasional combination with cancroid tumours are observed. The surgeons refer to these growths as "rodent ulcers." They are locally destructive, but have little tendency to generalize by metastasis.

As regards their situation, Boyd states that they do not occur at the junction of the skin and mucous membrane, but on parts such as the face, cheek, nose, eyelids, or ear. One may infer from this that the uncovered parts of skin, well supplied with non-follicular sebaceous glands, are particularly susceptible.

Gans attributes to this type of tumour a derivation from naevus-like structures. His detailed description agrees on the whole with what has already been mentioned. One point of particular interest is his statement that 5 per cent. of cases are combinations of the basal cell and spinous cell types. He maintains that the absence of keratinization is not an absolute characteristic of the basal-cell carcinoma. Some of these do show a tendency to horn formation, which is probably consistent with the degree of cell differentiation of that particular tumour. It has been observed that after X-ray therapy, basocellular carcinoma have sometimes given rise to metastases in the regional lymphatic glands. These metastases proved to be of the hornifying squamous cell type. He, however, draws attention to the fact that atypical giant cells with numerous nuclei and mitoses, or various other degenerative processes, are not as a rule seen in a basal cell-carcinoma.

For the sake of comparison, one might also consider the acuminate condylomata of man. Gans describes these as types of cauliflower, wart-like growths, usually of the external genitalia. They are at first single, but soon increase to large numbers when certain peculiar conditions of moisture and maceration favour their development (anal, genital, and mouth regions). Their situation, as well as their much-branched structure, renders them particularly favourable for the growth of all sorts of micro-organisms. The widening of blood-vessels and lymph spaces also facilitate the oozing out of exudate, which, together with secretions, forms a suitable pabulum for such organisms. The bacteria and spirilles, which are easily demonstrable in the foul-smelling secretion, cannot be regarded as causative agents. These growths are believed to originate from the spinose layer of the epidermis. They are, however, different from ordinary warts or papillomata, in that there is no thickening nor keratinization of the epidermal covering. It is thought that condylomata result from the action of a virus. Apart, therefore, from a certain outward resemblance, there is no reason to think that the tumours in Angora goats are in any way related or similar to the acuminate condylomata of man.

MORPHOLOGY AND PIGMENTATION.

Turning now to the tumours of Angora goats in the sixteen cases studied, there is undoubted evidence that they are of epidermal origin. In spite of their varied situations and certain other points of difference, they give the impression of belonging to the same group or at most to two allied groups of epithelial tumours—on the one hand, the undifferentiated, uniform, epithelial cell tumour, the *basal-cell carcinoma,* and on the other, the branching strands or solid masses of spinose or squamous cells with pearl-nest formation, i.e. the *spinous-cell carcinoma or cancroid.* Between these two extremes are cases showing a *combination* of the two types side by side in the same tumour, and others in which the state of differentiation of the cells seems in a *transitory stage* between the two types. Even amongst the simple basal-cell carcinoma, there are differences in appearances, both clinically and histologically. All have this in common, however, that they are composed of undifferentiated cells such as are seen in the lower layers of the epidermis. These cells may be round, polygonal, dendritic, elongated, or spindle-shaped. Only one type, however, is usually seen in any one tumour, so that the parenchyma has always a very uniform appearance. The arrangement of the cells differs also in different tumours. In some the cells are closely packed together with very little stroma, as in a sarcoma. In others a loose network is formed with the cells apparently well spaced out. In others again the cells show a more or less distinct arrangement into rows or strings, sometimes branching out in arborescent fashion, folded to simulate crypts or tubules, interwoven, giving a lace-like appearance, or fused to form solid strands.

Two out of the eight baso-cellular carcinomata, i.e. 17296 and 17297, showed extensive metastases, although there was definitely no sign of keratinization. The tumour cells in these cases showed numerous mitoses, a significant difference from the remaining non-metastasizing types of baso-cellular carcinomata. It is interesting to note also that Miescher mentions the frequent absence of mitosis in melanomata. He also states that the metastases from skin melanomata show the same characteristics as the primary growth. In cases 17296 and 17297 the metastases examined showed very little or no pigmentation, as compared to the mother tumour. Is it perhaps due to the protection of these parts from light that the cells are not stimulated to pigment formation? It seems that the younger tumours of this type, i.e. those still of small size and known to have recently developed, e.g. No. 7256 A, No. 7256 D, have usually the most undifferentiated and uniform structure. The older and larger tumours tend to malignancy and cancroid formation, e.g. Nos. 17293, 17298, 17296, 17297. In fact, in some cases of the combined basal and spinous cell carcinoma, the latter part developed secondary to the former, e.g. No. 14771.

It is also possible and even probable that those tumours diagnosed as spino-cellular carcinoma, e.g. Nos. 17293, 17298, and 7256 F, started as baso-cellular carcinomata. The spinous-cell part may have developed later, and then overgrown the original basal cell tumour completely.

The combined forms are those in which both basal and spinous cells types are present side by side, e.g. Nos. 14771, 14773, 21957.

The two types do not blend but are usually sharply demarcated from each other, and further, even when the basal cell part is pigmented, the spinous-cell portion just next to it remains free of pigment, e.g. Nos. 14771 and 7256 C.

The mixed or transition forms that are mentioned above represent a type such as No. 17294, in which there seems to be a blending or transition between the basal cell and the kenatinizing squamous cell One might almost suggest in seeing this and other similar tumours that by a process of evolution or differentiation the basal cells are gradually changing their ordinary appearance and acquiring the power of keratinization. Such modifications are not unknown in other forms of neoplasms, e.g. adenoma to carcinoma.

The question of melanin pigmentation in these tumours is a very interesting one. Some of the growths are totally devoid of pigment, while others show various grades of pigmentation. Macroscopically the intensity of pigmentation is reflected by the colour, which may vary through uniform or mottled shades of pinkish grey to pitch black. In section the concentration of melanin also varies widely. In some tumours only a few isolated epithelial cells contain granules of melanin. In others practically every cell shows the presence of fine granules diffusely scattered in the cytoplasms. In some cases the pigment may be confined to cells in one part of the tumour only. As a rule, pigment is most evident in those parts of the tumour which adjoin or run into the epidermis. That is to say, in those parts in which the cells of the basal layer show a peculiar " loosened " or " shelled out " appearance as they merge into the tumour tissue.

It has already been pointed out that the normal skin of Angora goats in the less hairy parts, such as the perineum and ears, may either be unpigmented (pink to the naked eye) or uniformly pigmented (tan or light brown to the naked eye). In addition, black or greyish black patches or maculae are very often present in these regions. (See Fig. 45.) These might be compared to the pigmented naevi of man.

A great deal of work has been done on the development of melanomata from such naevi. The " Abtropfungsprozess " of Unna and " Ségregation " of Darier, quoted by Miescher, are views on this which seem firmly established and have been supported by many other workers. The principle involved is briefly a loosening of " nests " of germinal cells from the basal layer of the naevus. These cell-nests then sink down and multiply in the corium or subcutis, thus giving rise to a melanoma. Miescher believes that there exist types of non-pigmented naevi, from which the non-pigmented epitheliomata are probably derived. The question arises, therefore, what is the difference between a basal-cell carcinoma and a melanoma or melanocarcinoma?

CLASSIFICATION.

In morphology, pigmentation, derivation, and other aspects, there appears, according to various workers, to be as many differences between the basal-cell carcinomata, or the melanomata themselves, as between the two groups. It seems thus that, until grounds for diffenrentiating the two are better defined, diagnosis in favour of one or the other must remain a matter of personal opinion.

After all, whether the mother cells are derived from the epidermis, including pigmetented and non-pigmented naevi, or from the skin appendages, they are all of the basal cell type. The resultant tumours must, therefore, within the ordinary range of variance, have a similar structure. Although pigmented tumours have not, as a rule, been considered as basal-cell carcinomata, there is no reason why, under certain circumstances, they could not be so termed.

It seems doubtful in any case whether pigmentation alone can be used as a differential criterion between the two. Furthermore, until the nature of naevi is better understood, one does not feel justified in assuming that the origin of tumours from them is sufficient reason to class them apart.

It has been stated by Wells (1925) that cells which do not normally form melanin, probably do not acquire this power in pathological conditions. This is, therefore, a very strong argument in favour of the basal cell origin of these tumours, since it is now a fairly well-established fact, from the work of Bloch and others, that this is the layer normally concerned with melanin production. (Owing to lack of the necessary reagents, it was unfortunately impossible to apply the Dopa oxydase reaction to these tumours.) The above statement of Wells leads one to believe that pigmentation in skin tumours is a secondary characteristic and depends entirely on the pre-existence of the melanin-producing capacity in the basal cells from which the tumour takes origin. A point of particular interest in this connection is that the spinous-cell carcinomata, in the cases studied, never contained any pigment. Even in the combined forms the pigment was sharply localized to the basal cell portion of the tumour. As far as can be ascertained, spinous-cell carcinomata in general do not contain melanin. This fact seems to support the view that the spinous-cell carcinoma takes its origin from the more differentiated layer of prickle cells, which, under normal conditions in the skin, are not capable of producing melanin. The spinous-cell carcinoma in the goat tumours must, therefore, be regarded as a separate tumour, which possibly develops as a sequel to the irritation due to the primary basal cell tumour. The " mixed form " can then be classed either as atypical cancroids or as keratinizing basal-cell carcinomata. For the latter Krompecher has already made provision in his classification.

The basal cell tumours described could have been called Melano-carcinomata and Amelano-carcinomata respectively. Such terms are vague and, moreover, might lead to confusion. The word melanoma in veterinary practice is often loosely applied to any melanotic tumour, and very often to melanosarcoma. For this reason the term basal-cell carcinoma has been used throughout to designate all the undifferentiated cell tumours of epidermal origin found in the Angora goat.

Gans gives Krompecher's classification of basal cell tumours as follows:—

Carcinoma baso-cellulare solidum.
Carcinoma baso-cellulare adenoides.
Carcinoma baso-cellulare parakeratodes.
Carcinoma baso-cellulare cysticum.
Carcinoma baso-cellulare hyalinicum.
Carcinoma baso-cellulare myxomatodes.

The basal cell tumours studied in this paper would thus fall in part under the first three subdivisions of the above classification. No provision is made for pigmented tumours. It remains, therefore, to be proved, by further detailed study of their finer morphology and genesis, whether a further subdivision to include them is justified or not.

PREDISPOSITION.

The etiology of cancer has so far baffled all attempts to elucidate it. The vast amount of work done in this connection has, however, not been altogether fruitless. The relationship between certain " predisposing factors " and the incidence of cancer has been closely studied, with the result that many aspects of the question are now better understood. Experiments and observations have thus far yielded overwhelming evidence in support of Virchow's Irritation theory, which to-day can practically be accepted as proved (Lewin, 1928). This leads one naturally to examine shortly the factors most likely to be involved in regard to the skin cancer now under consideration.

ENDOGENOUS FACTORS.

Breed.—It is evident that the Angora goat as a breed is much more susceptible, or less resistant, to skin cancer than the native or Boer goat under similar environments. Points of difference between these breeds might, therefore, yield a clue as to the reasons for such decreased resistance or increased susceptibility.

Heredity, Inbreeding, Constitution.—The Angora, as has already been stated, is a highly specialized animal for the production of mohair. The organ concerned most in this specialization is the skin. Inbreeding has been, and is, frequently resorted to, in order to accentuate certain desirable qualities in the skin and hair. In doing this, it is possible that a latent, inherent predisposition to cancer has also been accentuated throughout the breed. On the other hand, it may be that only certain strains transmit the predisposition. For the above reasons, also, the constitution of the Angora may have been weakened. As a result of her extensive work with mice, Slye claims to have proved that the disposition to cancer is hereditary, but that resistance is dominant over susceptibility. Little (1928), however, does not accept this theory, that cancer susceptibility is a simple mendelian recessive. Should breeding experiments of affected large animals be contemplated, the Angora goat would probably prove a suitable subject, on account of the relative frequency of spontaneous cancer.

Age.—Angora goats, on account of the mohair they produce, are probably kept alive longer than other breeds, which are usually destroyed for human consumption. Senile degenerative processes of skin and other pre-cancerous changes associated with age, have not yet been studied.

Sex.—It is peculiar that nearly all animals affected are females. No explanation for this can be given at this stage.

ANATOMICAL DIFFERENCES—PIGMENTATION.

Amongst others, one might mention the thinner and more delicate skin, the long hair, and the greatly developed skin glands under tail. On account of the configuration of perineum, secretions, excretions, and dirt are liable to stagnate, decompose, and act as irritant and macerating agents in this region.

Pigmentation of the skin is one of the most striking differences between the Angora and the native goat. The latter have invariably a very heavily pigmented and tough skin. According to Wells (1925), the function of melanin is that of protection against light, especially the violet and ultra-violet rays. This protection is at least in part responsible for the relative infrequency of skin cancer in the coloured human races (Wells, 1925). It is significant also that the parts of the skin mostly affected are those not protected by long hair. In Merino sheep, which also have a non-pigmented skin, the cancers thus far noted also affected the face and ears. The part played by the pigmented patches or naevi is not yet understood.

EXOGENOUS FACTORS.

Change of Habitat.—Although not native to this country, the Angora has become fairly well acclimatized. This, however, does not mean that climatic and other influences have no action on the susceptibility to cancer.

Irritants.—Tumours in Angora goats can only rarely be traced to traumatic injuries, in spite of the fact that the latter are frequent, ear-marking, horning, e.g. 14505. One type of injury which seems to give rise to tumours more frequently than others is the fracture of a horn at its base, e.g. 17292, 17296.

As regards chemical and physical irritants, very little is known. Sunlight, and maceration by decomposing secretion, have already been mentioned. It is interesting to note that the Angora goat is highly refractory to the carcinogenic action of coal-tar. (See Experiment 10.)

Other possibilities have been suggested as starting points for tumours, e.g. tick bites with resulting wounds and scratching.

"*Piles.*"—These anal tumours have often been called "bleeding piles" by farmers. The possibility of these tumours starting from haemorrhoids was not overlooked. This can, however, definitely be ruled out. Haemorrhoids, according to Fischer, are enlarged varices on the venous plexus either inside or outside the anal sphincter, under the mucous membrane of the anus. The tumours described are definitely associated with the epidermis. Haemorrhoids are practically unknown in the lower animals, and, further, even in man, it is very seldom that they give rise to any malignant tumour.

In conclusion, it is very difficult to pick out any one or more of the above factors and attach to it more importance than to others. There may be other factors at play of which nothing is known. In all likelihood it is to the combined influence of all or most of these factors that the neoplasms are to be ascribed.

V. SUMMARY AND CONCLUSIONS.

1. The South African Angora goat is considered briefly in regard to its origin, appearance, and habitat. Special attention is paid to the histology of the normal skin of those parts usually affected with cancer.

2. The occurrence of a type of skin gland in the depth of the subcutis of the perineal region is recorded. It is not mentioned in textbooks on comparative histology. It is suggested that it may be a type of apocrine sweat gland.

3. Attention is drawn to the irregular distribution of melanin pigment in the skin, and the frequent occurrence of naevus-like pigmented patches.

4. Skin cancer of the South African Angora goat is described apparently for the first time.

5. Its incidence is relatively high, especially when compared with the native and Boer goats in this country, in which cancer is practically unknown.

6. Females are by far the most frequently affected.

7. The predilection seat of development is the perineum (anus and vulva). The ears, horn stumps, and the rest of the skin are also occasionally affected.

8. The material studied is derived from 16 cases. Thirteen were tumours of the perineum, one of the ear, one of the horn, and one of the neck.

9. According to histological appearances, these tumours were divided into three groups, viz., (i) the undifferentiated epithelial cell tumour, or basal-cell carcinoma; (ii) the differentiated cell tumour or spinous-cell carcinoma; (iii) mixed or combined forms of both.

10. In most cases of basal-cell carcinoma, the relationship of the tumour cells to the basal cells of the epidermis can be seen clearly. Pigmentation is closely associated with this region.

11. Evidence from two cases, 14774 and 9983, seems to point to the sebaceous gland as the origin of at least some of these tumours.

12. As the tumour grows, the proliferating basal layer remains continuous with that of epidermis. In this way pigment-forming cells as they occur in epidermis become incorporated and proliferate in the tumour.

13. Spinous-cell carcinomata seem to originate from already differentiated epithelial cells, as they have not the property of pigment formation.

14. It is suggested that these cancroids arise sometimes secondarily, as the result of irritation due to pre-existing basal cell tumours.

15. The spirochaetes and bacteria found on the tumours are secondary saprophytes and cannot be regarded as the cause of these tumours.

16. Goats suffering from cancer often become fly-struck. The fly larvae undoubtedly contribute additional suffering to the animal and may hasten death or metastasis.

17. Surgical treatment is fairly simple in the early stages, and in our hands has been entirely successful.

18. On account of the small individual value of the goat and the difficulty in obtaining veterinary assistance in this country, it is advisable at this stage to encourage culling of all affected animals.

19. Several attempts at transmission by contact, inoculation, and transplantation failed.

20. The Angora goat is highly refractory to the carcinogenic action of coal-tar.

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VII. PROTOCOLS.

In order not to encumber the text matter with rather lengthy descriptions of each individual case, the clinical observations, post-mortem and histological findings are gathered together in protocol form under this heading. The cases are dealt with in the order in which they are arranged in the comparative table given above. A short comment on the diagnosis will also be found at the end of each description.

GOAT No. 14771.

Clinical Observations.

Angora she-goat, full-mouth, in fair condition. 6/9/26: In the recess under tail an irregular lobulated tumour is found, measuring roughly 3.25 × 2.5 × 1 cm. It is of a pinkish red colour, soft in consistence, giving one the impression of exuberant granulations. It is extremely vascular and bleeds easily on manipulation. It is covered by slimy greyish exudate, mixed with blood and in parts tending to form crusts. Attachment is by a short stalk to the right and above the anus, and involves only the skin, so that the tumour is freely movable over subcutaneous tissues. The anal opening is just covered by the edge of the tumour, but is otherwise quite patent, and defecation is not hampered in any way. The vulva is unaffected. The skin around vulva is pigmented in patches (melanosis).

22/4/27: Tumour has become rounded in shape, roughly the size of a walnut, it is still deep pink, soft, and glistening, and covered by slimy material soiling and matting the breech hair. This is further increased by a purulent discharge from the vulva. A small elevation of skin is noticed near the dorsal commissure of vulva; this involves one of the black pigmented patches of skin mentioned above and is kept in a practically constant state of moistness by the sticky exudate running down from tumour.

Smear preparations made from the surface of the tumour show a rich mixed bacterial flora, with which is mixed numbers of spirochaetes and also fusiform organisms.

16.5.27: The tumour tends to spread out into a mushroom-like shape. It is still lobulated and pinkish red on the surface, like granulation tissue, and bleeds as easily when touched. The swelling above vulva has extended downwards considerably and involves the whole of both lips of vulva. The whole vulva shows diffuse swelling and is covered by slimy exudate. The most prominent part is near the dorsal commissure. The exudate has dried to tenacious crust, firmly adherent to underlying tissue. When this is removed by force, a bleeding, rough, black ulcer, with raised swollen edges, is exposed. A smear from the surface of this ulcer shows that the black colour is due to a greenish black, granular pigment present in large amounts and partly lying freely, partly hoarded by large mononuclear cells.

Defecation is somewhat painful, but takes place with little difficulty. The animal, however, appears to suffer great pain when manipulated in region of perineum and tail. Urination is normal and unhampered.

2.6.27: Practically the same, the vulva is covered by a hard tenacious crust which causes distortion of underlying tissues. There is a purulent discharge from vulva and from beneath the crust. Bleeding takes place when the crust is removed.

23.6.27: Anal tumour is about the same size, pinkish grey colour, covered by slimy stinking matter. The vulva shows the same swollen distorted appearance, with tenacious crust and black bleeding purulent ulcer beneath. The structure beneath the scab is moist and soft and gives the impression of being spongy.

4.7.27: Tumours still in the same state, the black vulvar tumour if anything smaller. Smears from surface show numerous spirochaetes, fusiform organisms, and a varied mixture of other bacteria. Large quantity of greenish-black pigment, either free or hoarded in mononuclear cells from surface of black tumour.

9.9.27: Size and appearance the same. The tumours are covered by the moist sticky exudate, which, mixed with caked blood, etc., matts and soils the perineum and surrounding hair.

14.9.27: This animal was given 1.0 g. pot. iodide daily to see what effects this drug would have on the tumour.

27.9.27: Tumour somewhat reduced in size, now measures only 3 cm. in its greatest diameter.

5.10.27: Dose of pot. iodide increased to 2 g. daily; no change can be noted in tumour above anus, but the black vulvar tumour is somewhat decreased in size and covered by a scab. There is a slight watery discharge from the eyes, but the appetite and condition of the animal does not seem to suffer.

7.10.27: Tumours appear to decrease in size. The black vulvar tumour has the appearance of drying up, and is covered by thick tenacious scab. This can only be removed with difficulty and causes great pain. Spirochaetes and bacteria still present on the surface of tumours.

13.10.27: Eyes inflamed and show a slight purulent conjunctivitis. There is a serous discharge from nostrils and dribbling of saliva from the mouth (iodism). Dosing with potassium iodide discontinued. Except for the somewhat drier appearance and slightly decreased size, there is no change in tumours.

15.10.27: Animal has a distinctly dejected appearance and stands hunched up, hardly feeds at all. Condition noticeably poorer, purulent conjunctivitis worse.

17.10.27: Emaciated condition, hunched up and listless, conjunctivitis, and dribbling from mouth.

21.10.27: Very much weaker, emaciated, does not feed. Seropurulent discharge from eyes and nostrils.

22.10.27: Lying down, unable to rise.

24.10.27: Died during the night.

Post-mortem Findings.

Cachexia, purulent conjunctivitis, catarrhal rhinitis, catarrhal enteritis. The tumour under tail measures 2 × 1.5 × 0.5 cm. It is pinkish red in colour, smooth and covered with slimy greyish exudate.

The tumour over dorsal commissure of vulva is pitch-black in colour, measures roughly 1 × 0.5 cm., is fairly hard, and covered by tough scab. The swelling extends downwards on either side of vulva. The rest of skin of vulva is black in patches, pigmentation extending inwards along the mucous membrane for 3 cm. Death in this case is probably due to potassium iodide poisoning.

Histological Findings (Specimen No. 7447).

Anal Tumour.—Under low magnification the appearance is as follows: On the one side the normal epidermis with all its skin glands becomes abruptly replaced by a very vascular tissue with ragged surface, indented by fissures which throw it into rounded, irregular lobes. In places the surface is covered by a thick layer of necrotic matter. This tissue on the other side becomes continuous with the epithelium of the anus. At this junction there is a circumscribed, thickened, convoluted, solid nest of basal cells connected by a small bridge to the anal epithelium. The central cells are not quite differentiated into squamous cells, and there is no indication of keratinization. This nest marks the end of the epithelium of anus. The vascular tissue mentioned above forms the boundary of the tumour proper. In between the numerous small blood-vessels and capillaries engorged with blood, there is a large amount of inflammatory exudate cells mostly neutrophiles and lymphocytes. Amongst this there stands out clearly solid strands and nests of epithelial cells. These are mostly arranged in the form of strings of single cells bent to form crypt-like structures, or growing into closely packed masses, mostly at the bottom of the fissures between lobes. These cells show no mitotic figures, but evidence of rapid amitotic division is not lacking, as cells with two or more nuclei are frequent, and many nuclei have several nucleoli. These cells have not the regular uniform appearance seen in 7256 A, for instance, but show a slight tendency towards the squamous type, i.e. lighter staining and polygonal shape. There are numerous swollen faintly staining cells, with dark chromatic granules in place of nucleus, the so-called " cancer parasites," but no definite centres of keratinization are present.

DIAGNOSIS.

Basal-cell carcinoma (adenoid type).

Vulvar Tumour.—The vulvar tumour is practically continuous with the anal part above, but is quite distinct in that it is pigmented and affects only the vulva and its mucosa. The mucosa of vulva for about 2-3 cm. is of a pitch-black, patchy colour, but is otherwise smooth. The skin of vulva is somewhat swollen and shows loss of substance over a small area. This elevated portion is covered by a layer of necrotic matter, mixed with a large amount of pigment. The underlying tissue shows numerous small blood-vessels and capillaries. In between these, and penetrating a small distance from surface only, there are numerous nests of epithelial cells of the squamous type, surrounded by germinal layer in more or less the typical cancroid fashion. These nests are separated by large numbers of inflammatory cells (lymphocytes, neutrophiles). There is a considerable amount of black pigment (melanin) sometimes associated with the basal cells, but mostly in dark clumps in the interstitum, i.e. in connective tissue cells. Pigment was never seen within the horny centres of the above epithelial nests. From the entrance of the vulva, and for a short distance along the mucosa, the structure changes. The horny epithelial nests are replaced by foci of uniform large polygonal cells, all containing melanin in the form of diffuse fine granules. Their structure, which is undoubtedly epithelial, is thus not obscured. They are not too closely packed together, and have the same staining properties and shape as the basal cells of epidermis. The only difference is that they appear much larger and the nuclei fainter. The pigment is in the form of a fine dust-like mantle around the nucleus, whereas in the deeper tissues the pigment is hoarded, in the form of very compact and dark clumps, in connective tissue cells (histiocytes). The thickness of this pigmented layer of basal cells varies. In parts it is in the form of round circumscribed foci partly covered over by flat epithelium, but towards the deeper parts of the vulva the layer becomes thinner, and gradually merges into normal mucosa. The pigmentation there appears to be due entirely to melanin hoarded in the deeper connective tissue cells.

DIAGNOSIS.

Pigmented baso-cellular carcinoma with squamous cell (cancroid) tendencies in parts.

COMMENTS.

This is a good example of the mixed type of carcinoma often found. The anal tumour is a non-pigmented basal-cell carcinoma. The vulvar tumour is a deeply pigmented variety of the same, with the formation of cancroid "pearl nests" or keratinization centres. Several points of interest will be noted in this case.

1. The primary tumour was presumably a basal-cell carcinoma of the anus, without apparent pigmentation.

2. There was a pre-existing melanotic patch of skin at the dorsal commissure of vulva.

3. On this portion of skin a pitch-black tumour subsequently developed; partly raised and indurated, partly ulcerating and spongy in character.

4. Histologically, therefore, three forms of epithelial new-growths present themselves, i.e. pigmented and non-pigmented basal-cell types, the former in close proximity with keratinizing epithelial strands of the cancroid type.

5. The purely cancroid parts of the tumour do not contain pigment.

6. The cancroid vulvar tumour developed a considerable time after the "primary" anal tumour.

In this connection, does the continual macerating effect of the exudate which runs off the primary tumour play a part in the production of the cancroid, which appears to have developed subsequently? It is now fairly widely accepted that the type of neoplasm which develops as a result of chronic irritation, including tar cancer, is of the cancroid type. The question is a diffcult one to answer. There are so many possibilities that, beyond making the suggestion, one does not feel justified in forming an opinion.

GOAT No. 14772.

CLINICAL OBSERVATIONS.

20.8.26: Angora she-goat, aged (only stumps of incisors left). Condition fair. The perineum presents a dark red, rounded tumour, hanging by a short stalk from the lower, left aspect of the anus. It is granulomatous in nature, soft, covered by a moist, sticky exudate, and bleeds very easily when manipulated. Its size roughly is 2.5 cm. wide, 1.5 cm. high, 1 cm. thick. The anus and vulva openings are unaffected, so that defecation and urination are not interfered with. Just above the vulva the perineal skin shows a pigmented patch (melanosis). On this date the whole of the tumour was excised surgically for histological study. It was found to be attached only to the skin and did not penetrate or involve the underlying tissues to any extent; the stalk being about 1 cm. in thickness. The operation wound was treated antiseptically and was completely healed up in 15 days. This animal was then kept without any further interference, being only examined from time to time. In this way it was observed that at times the cheesy matter accumulating beneath the tail would cake into small crusts which, on being scratched off, often left a small bleeding erosion of skin. These, however, healed up repeatedly and never showed a tendency to develop further.

This animal was killed on account of old age and poverty on 27.9.28, i.e. about 25 months after removal of tumour.

Post-mortem Findings.

Emaciation, anaemia (due evidently to malnutrition). A few specimens of *Haemonchus contortus* in stomach. Old encapsulated abscesses in lungs. The post-mortem examination otherwise negative. The perineal skin was somewhat roughened and showed small elevations above anus, but otherwise the skin was fairly soft and pliable still.

Histological Findings (Specimen No. 6189).

Small almond-shaped tumour removed surgically on 20.8.26. There is a central core of fibrous tissue continuous at the base of tumour with the corium. It ramifies in fan-shaped fashion to form a delicate stroma which supports a closely packed, very uniform mass of epithelial cells. They have large, round, or oval nuclei with one or more nucleoli, which are rather poor in chromatin. The cytoplasm seems to be absent in most cases, or nearly so. This apparently is due to its very delicate nature. Indeed, it seems that the very finely fibrillar substance often drawn out into shreds and elongated processes of various shapes in which the above nuclei are more or less embedded constitutes the protoplasmic part. This in life probably is very delicate and soft, so that it undergoes these changes during preparation of the sections. This substance is apparently gathered along the stroma in greater amounts, so that the cells lying away are separated by small empty spaces. The stroma has thus the appearance of trellis work, composed mostly of this finely fibrillar substance and tumour cells, amongst which young fibroblasts, round cells, neutrophiles, and blood capillaries can be seen. There are also numerous smaller cells with elongated, deeply chromatic nuclei, possibly of endothelial origin. Blood capillaries and smaller vessels are numerous, especially near the surface. In parts small haemorrhages can be seen near surface. These contain brownish pigment, like haemosiderin. No melanin could be seen in the whole of this tumour. Mitoses, if at all present, are very rare in the tumour cells. By following up the small amount of epidermis covering the stalk one side, one reaches a point where it merges into the tumour. There is a peculiar elongation of the germinal cells at this point, so that they appear to be loosened and " shelled out " into the tumour parenchyma. There is very little infiltration with inflammatory cells, considering that the whole tumour is covered only by a thin layer of necrotic exudate.

Diagnosis.

Baso-cellular carcinoma.

Histological Findings (Specimen No. 8481).

After the death of the animal, sections were prepared from various portions of the perineal skin. Particular attention was paid to those parts above anus which showed small elevations. The epidermis here appears much thinner than usual, as if atrophied, in parts being barely 3 to 4 cells thick. The sebaceous glands in this region are about as numerous as in the normal skin, but show this peculiarity, that the basal cells appear proliferated to form a dark staining, well-defined layer all round the central mass of fatty cells. In one place, what appears to have been a sebaceous gland from its circumscribed indented outline, causes a slight outward bulge in the epidermis. This round, elevated focus, between 1-2.5 mm. in diameter, consists practically of a solid mass of epithelial cells similar to those forming the basal layer of sebaceous glands. (See Fig. 33.) It is situated in the corium, but it is not visibly connected with the exterior as it is still covered by intact epidermis. The nuclei of these cells are fairly closely packed together and no fat-carrying gland cells are left. Serial sections were prepared from the remaining material and showed that the focus was more or less globular in outline. The structure of this focus certainly has a close resemblance to that seen in the closely packed round cell epithelial tumours, such as, for instance, the one removed previously from this animal and described above, and also No. 7256 A and 7256 D.

Comments.

The tumour removed surgically proved, on microscopic examination, to be a simple example of a non-pigmented basal-cell carcinoma. Twenty-five months later the animal was killed and portions of skin in the same region examined. To the naked eye a few small elevations of skin could be noted. The epidermis was thinner than usual (senile atrophy?). The elevations were due to small foci of closely packed epithelial cells resembling those seen lining the neighbouring sebaceous glands. It seems possible, therefore, that, by a process of proliferation, the basal cells of the sebaceous glands displace the fatty cells, without themselves undergoing fatty changes.

As growth continues the epidermis becomes pushed out by the enlarging focus and becomes thinner and thinner until it finally ulcerates. The epidermis is in direct contact with the underlying tumour cells at least near the base. The basal layer of the sebaceous gland and that of the epidermis are fundamentally continuous with each other, so that, allowing for displacements, the derivation of the tumour cells from the germinal layer should remain apparent in the young neoplasm.

This appears to be a reasonable explanation of the origin of basal-cell carcinoma and one that has already been suggested by various authors.

GOAT No. 14773.

Clinical Observations.

Angora she-goat, aged, incisors worn down to gums, condition fair.

6.9.26: On lifting the tail a large newgrowth measuring 5 by 4 cm. occupies the whole recess under the tail. It extends downwards between the tail folds on either side and covers the anus and the upper commissure of the vulva. It is attached above the anus over an area roughly 2.5 by 3 cm. Its thickness varies from 2.5 cm. above to 1 cm. below. The tumour is practically pitch-black in colour throughout. It is irregular in outline and presents a moderately lobulated appearance, with a deep fissure running across its surface obliquely. It bleeds very easily on the slightest manipulation. On account of the weight of the tumour hanging from the skin and underlying tissue, the anus is not closed properly, but its upper half overlaps the lower. Defecation and urination are not, however, interfered with, and there are no signs of collateral inflammation.

18.10.26: The tumour has increased in size, it now measures 12 by 8 by 2.5 cm.

19.11.26: This goat was killed by an intratracheal injection of chloroform and ether. The whole perineum was excised, together with attached tumour, with a view to utilizing parts of it for transplanting experiments, and for histological examination, respectively. (For transplantation and transmission, see Experiment No. 2.)

Histological Findings (Specimen No. 6377).

Under the lower-power lens this tumour presents a remarkably uniform structure, as is seen usually in a sarcoma. The cells are arranged in whorls according to the sweeping curves of the branching stroma coming out of the stalk. The surface is covered for the greater part by a necrotic layer of exudate and other cells. Directly under this is a zone conspicuous by the close proximity of blood-vessels and capillaries. From the small amount of connective tissue and the staining property of epithelial cells, it seems that active growth takes place in this region. Melanin is strewn throughout the tumour parenchyma. Under a higher magnification it is seen that the distribution of the pigment is very irregular. Many of the epithelial cells contain a varying amount of pigment, but some have none at all. On the other hand, a fair number of cells are packed so full of it that they appear as large black blotches in the field, and are often aggregated together in bunches. Such clumps are seen fairly frequently in the connective tissue stroma and in the deeper parts where epithelium is not usually found. Such heavily packed cells are probably connective tissue cells whose function is the hoarding of melanin. The stroma carries much more pigment than the tumour parenchyma. (See No. 7256 B.) Some of the melanin also lies free in the interstitial spaces, but of course this distribution may be accidental, due to spreading during the cutting of the section. There are very few mitotic figures in the tumour cells. In other respects the structure agrees with that of specimen No. 7256 B, C, D, etc. In the deeper subcutaneous tissue the tumour parenchyma is bounded off by a thick connective tissue capsule. In parts of the tumour, foci of somewhat different cells can be seen, they are slightly smaller, rounder, and stain a deep purple. They do not contain any pigment, but for the rest are arranged as the others.

In many places where the epidermis can be followed until it merges into the tumour, the typical " shelling out " effect of the altered basal or germinal cells into the tumour parenchyma can be clearly observed. The epidermis shows acanthosis to a marked extent, and in one place goes over abruptly into solid strands of squamous cells, closely packed together and having many foci of keratinization typical of cancroid horn-pearl formation. This part is limited in extent, and is separated by connective tissue from the rest of the tumour. It is, however, continuous with the acanthotic epidermis. It contains no pigment.

DIAGNOSIS.

Pigmented baso-cellular carcinoma, with a small spino-cellular carcinoma next to it. Combined form.

GOAT No. 17293.

CLINICAL OBSERVATIONS.

Angora she-goat, aged. Very poor condition.

17.6.27: A large pinkish tumour is found to occupy the whole of perineal depression below the tail. The tumour is about 5 by 4 cm. and attached by a very short stalk to a small area above the anus. The surface is nearly flat, but shows shallow fissures. The tumour is covered by a moist, sticky, greyish to brownish, foul-smelling matter, which causes a general soiling and matting of hair around the breech. The vulva is unaffected, although the anus is partly overlapped by the edge of the tumour; defecation does not seem to be much interfered with. When manipulated in region of tail, the animal evinces considerable tenderness, and the tumour bleeds very easily on the slightest handling.

Smear preparations from surface shows a rich and varied bacterial flora, but no spirochaetes.

9.7.27: Matting and soiling of hair around perineum accentuated, evidently due to increased exudate from tumour. The tumour itself shows on its left surface a sunken patch of greenish grey, softer substance (gangrene).

15.7.27: Foul-smelling and objectionable appearance, tumour largely gangrenous on surface.

In order to check this process, the tumour was cleaned with tincture of iodine and then coal-tar applied.

18.7.27: Gangrenous parts have sloughed off, leaving a relatively clean, granulomatous, pinkish tumour behind, a good deal smaller than before.

2.8.27: Greyish exudate has again increased and greenish patches of gangrene appear on surface of tumour. Rest of perineum moistened and macerating.

4.8.27: The dorsal commissure of vulva is swollen and is covered by semi-dry scabs. No spirochaetes can be found in smears made, although the bacterial flora is profuse.

8.8.27: Superficial gangrene of tumour present.

15.8.27: Profuse thick yellowish-grey stinking exudate, soiling the whole perineum and matting the hair at sides.

26.8.27: A large crop of fly larvae found all round and under tumour.

2.9.27: The tumour is considerably reduced in size, due to sloughing off of grangrenous tissue. Fly maggots are present and seem to cause much discomfort to animal. It keeps its tail down and flicks it continually, often stamps its feet, and shows uneasiness generally.

6.9.27: Most maggots have disappeared, tumour moist, sticky, and slightly necrotic. Superior commissure of vulva is much swollen, moist, and pink in colour, being continually smeared with exudate by action of tail.

13.9.27: Only a few large maggots left; there is ulceration on both anal folds of skin. Anus is practically covered over by edges of tumour, but defecation apparently not interfered with. Vulva moist and swollen, scabs adherent over the tumour.

15.9.27: Hair shorn off. Condition of animal is rather poor, and skin shows scaliness, somewhat resembling dandruff.

21.9.27: Dosed, from to-day, daily with Liquor-arsenicalis B.P., starting with 0.1 c.c. and gradually increasing to 0.4 c.c.

7.10.27: No change. Spirochaetes are found in the smear preparation from surface of tumour.

13.10.27: Discontinued arsenic. This had no effect on tumour, which is still as before. Animal is noticed to lie down a good deal.

15.10.27: Animal lies down in corners with head bent towards tail as if to ward off flies. These seem to cause it a great deal of worry.

19.10.27: Given potassium iodide 2 grams, liquor arsenicalis 0.4 c.c. daily from to-day. Tumour is about the same, covered by sticky, foul exudate and in parts with scabs.

25.10.27: Animal lying down a good deal and is in poor condition.

29.10.27: Slight inflammation of conjunctiva with watery discharge, discontinued the potassium iodide, but continued the liquor arsenicalis. The anal tumour has decreased slightly in size.

2.11.27: The tumour now measures 3.5 × 2 × 1.5 cm., the greatest length being transversely across from one side to the other. The perineal skin above vulva also much thickened and covered by scabs. Smear shows numerous spirochaetes, fusiform, and other bacteria. Increased daily dose of liquor arsenic to 0.6 c.c.

9.11.27: Slight greenish gangrenous areas on tumour, vulva swollen and moist, partly crusted over, and showing a few small prominences of skin, especially on left side. Given potassium iodide 2 grams and liquor arsenic.

10.11.27: Given potassium iodide with arsenic as before.

12.11.27: Do. Tumour gangrenous, with profuse foul-smelling and sticky exudate.

14.11.27: Necrotic area sloughed off, leaving a fairly clean granulation-like pink surface of tumour. Given potassium iodide and arsenic as before.

21.11.27: Potassium iodide has been discontinued, but the liquor arsenicalis daily dosing is proceeding. The animal is in poor condition, lies in corners and away from the others, seems extremely worried by flies settling on the tumour and often bites at perineum. It occasionally groans when defecating and appears very tender when tail is elevated or tumour touched in any way.

22.11.27: Liquor arsenicalis discontinued. Tumour shows no change. A slight sero-purulent discharge from the eyes (iodism).

28.11.27: Poor in condition and weak, lies down most of the time, but appetite still good.

13.12.27: Partial necrosis of tumour. Vulva moist. macerated, and swollen in appearance. There is yellowish-grey excrescence on the lower part of the left vulva lips. This is covered by crusts and is about the size of a pea. The lymphatic supramammary glands show no enlargement on palpation.

30.12.27: Emaciated and weak, unable to follow other goats. Small protuberance on left lip of vulva grows very slowly and is usually covered by crusts.

14.1.28: The anal tumour assumes a flattened, pink, cauliflower-like appearance, occupying the whole of space under tail and partly covering anus. The vulva is swollen and moistened by the exudate, and the protuberance on the left side has grown to the size of a hazel-nut. The animal shows much uneasiness and scratches frequently at perineum, although no maggots are visible.

30.1.28: Fly larvae present in large numbers, tumour is somewhat enlarged. The whole perineum has a repulsive appearance owing to foul-smelling brownish exudate soiling all neighbouring tissues. The maggots swarm all over and are eating into the tumour. Goat is still very sensitive and bleats weakly when handled, e.g. lifting tail. It is emaciated in spite of fair appetite. Continual irritation is shown by running around and flicking tail, stamping feet, biting at tumour and lying down in corners. etc.

25.2.28: Tumour slightly decreased in size owing to necrosis and burrowing action of maggots. Smaller tumour of vulva is the size of a hazel-nut and has a greyish, granulated rough surface, being covered by sticky exudates and scabs. The other side of vulva is swollen.

15.3.28: Animal is so emaciated and weak that it lies down most of time. The anal tumour has a very irregular cauliflower-like surface, covered by slimy exudate and necrotic matter.

27.3.28: Tumour enlarged somewhat, tends to bulge out the tail folds on either side. The whole mass is not thicker than 2 cm., being pitted and fissured in the centre with a raised swollen border all round. The anus is exposed and is fringed by a ragged edge of granulomatous growths. Animal evinces considerable pain in defecation, it groans and strains, and only a few pellets are voided at a time. The whole vulva is swollen and macerated, the lower part of left lip carries a growth with rough, greyish, granular surface about 2.5 cm. in diameter.

11.4.28: The right supramammary gland shows a decided enlargement, it feels hard and about the size of a walnut. The left one is about the size of a french bean and softer on palpation.

18.5.28: Right horn broken off short, frontal sinus teeming with fly larvae and necrotic matter, treated and tarred. The anal tumour is one large, ragged mass of stinking, partly necrotic, slime-covered tissue. It is roughly rounded in outline and occupies the whole space between tail folds. It is shallow and contains deep fissures and burrows which are filled with maggots. The edge all round is ragged, swollen, and elevated, so that the whole tumour may now be regarded as a huge ulcer, which is confluent with the tumour on vulva. Faeces now seem to emerge from a fissure on the bed of the ulcer. The anus has been more or less completely broken down. Urination takes place without difficulty. The animal strains and groans whenever it passes faeces, and bleats whenever any of the affected parts are handled. The surrounding hair is much soiled and matted together by a profuse, slimy, stinking brownish exudate, to which dirt sticks, and forms crusts in many places. The animal is very weak, but still shows signs of great distress from irritation of perineum and tumour. The right supramammary lymphatic gland, on palpation, feels the size of a walnut and is fairly firm. The left is still the size of a large bean.

23.5.28: Found dead in the morning.

Post-mortem Findings.

Extreme cachexia and anaemia. Fractured right horn with necrosis of frontal sinus. Sarcosporidia in oesophagus; hydropericardium. Large ulcerating tumour roughly circular in outline, with deeply fissured and pitted bed. The raised and much-swollen borders cause great distortion of vulva to the right side. Its diameter is 7 cm. and it extends from the recess under the tail, down to the dorsal commissure of the vulva, and then down the left lip at the lower part of which is a greyish, rough protuberance, covered by a scab. The deep crevices in the bed of ulcer are filled with fly larvae which are actively eating away deeper into tissues. The anus has completely disappeared, the rectum opening directly on to the floor of ulcer. The vulva is patent in spite of distortion due to swelling.

The right supramammary gland shows at one side a small metastatic focus about 1.5 cm. in diameter. There is a large abscess in the pelvic region outside the rectal wall. This contains a mass of thick yellowish pus.

Histological Findings (Specimen No. 8059).

Section of this tumour at its junction with normal skin shows various transitory stages very clearly. The epidermis very abruptly becomes enormously thickened, sending down long blunt and sharper processes into corium. These consist of flat prickle cells. Almost immediately, marked cornification takes place. The most superficial areas show large nests of keratinizing flat cells, with nuclei still more or less retaining their outline. The surrounding flat cells are paler in colour with vesicular nuclei and usually flattened concentrically around the pearls. This tendency to horn formation is marked even in cells fairly deep near the basal layer. There are very numerous cells, isolated or in groups of two or three, which show the peculiar swelling out and hyalinization of cytoplasm and the disintegration of nucleus to dark chromatic masses or granules. In parts this leads to the formation of most weird-shaped giant cells. (See figure.) The cytoplasm reaches an enormous size, stains faintly, but is not quite hyalinized. The nuclei show rapid division to form a conglomerate of several, or else they disintegrate to form large dust-like chromatin masses enclosed as it were in a delicate membrane.

Further towards the centre of the tumour the pearl-nest formation become less frequent, the squamous cell strands become more solid and branch frequently in all directions, penetrating to varying depths in the intermuscular spaces. There is a strong supporting connective tissue stroma. The surface is covered by an irregular necrotic layer of tumour cells and exudate, in parts showing great excavations or fissures. The stroma is more or less infiltrated with polymorph neutrophiles, often forming small abscesses. Skin gland are totally absent from the tissue below the tumour. The supramammary gland shows large dense foci of typical flat squamous epithelial cells with centres of keratinization (cancroid).

Diagnosis.

Spino-cellular carcinoma (cancroid) with metastasis in supramammary lymphatic gland.

GOAT No. 17294.

Clinical Observations.

Angora she-goat, aged, poor in condition.

17.6.27: A small tumour 2 × 1 cm. is attached above and to the left of anus. It is covered by a thick crust of dry exudate and dirt, which is securely adherent to the underlying tissues. The surrounding skin and hair are quite dry and fairly clean. A smear from surface scraping was made, but showed only a small number of bacteria. No spirochaetes were present.

30.6.27: The thick crusts covering tumour were removed with some difficulty, and left a raw bleeding protuberance.

9.7.27: Thick scab firmly adherent to the tumour, when removed, leaves a bleeding protuberance, each time smaller than before. A thick layer of tumour seems to come off with the scab on removal.

22.7.27: Hard circumscribed crust covers just a small raised nodule about 1 cm. in diameter, the remainder of original tumour. There are about two or three small excrescences covered by thick tenacious crusts next to it. On removal small bleeding excoriations are left.

2.8.27: The same elevations of varying sizes, covered by dry scabs, are present. There is no tendency to produce moist exudate; the covering scabs are partly of a horny nature.

15.9.27: Animal shorn; condition has improved markedly, result of advanced state of pregnancy. Perineum the same.

23.9.27: The elevated areas at perineum are reduced in size and covered by small crusts only. The tumour has disappeared completely, although the site still shows a bleeding excoriation when the scab is removed.

5.10.27: Goat has given birth to one kid during the night. This kid is very weak, and is unable to feed.

13.10.27: Kid feeds fairly well after some difficulty had been experienced in getting it to learn to stand and suckle. Its legs are very weak, and inclined to give way under the body, spreading out at right angles to body on either side. The goat itself shows a few small pustules on perineum, which is partly covered by brownish discharge from vulva. The recess above anus shows a few small elevations of skin not covered by crusts, but presenting a superficial exoriation.

19.10.27: Crusts have reappeared on the small raised excoriations. The perineum otherwise shows a few scattered pustules extending down the escutcheon to udder.

29.10.27: Pustules healed up, few small dry crusts above anus.

2.11.27: Photographed perineun after removing the dry crusts and dirt, leaving the small excoriations bare.

13.12.27: The crusts on being left undisturbed thicken considerably and even become partly horny and pendulous, being very firmly adherent to underlying skin. Small surface erosions of skin can be seen at various places near anus and vulva.

3.1.28: Pendulous crusts have fallen off, leaving small ulcers which heal partly and form new scabs.

15.3.28: Animal in good condition, small dry scabs as before covering little superficial excoriations.

21.6.28: One of the largest scabs has grown out, forming a long horny claw-like process 1 cm. in length. When forcibly removed this leaves a small bleeding papilla. On the left side of anus near above papilla, there is a blackish grey flat crust raised above surface and about 1 cm. in diameter. This when removed leaves a raised, ragged, bleeding surface.

15.9.28: The above tumour or raised ulcer has enlarged to 1.5 cm. in diameter. It is not covered by crusts, but presents a rough semi-dry surface.

24.9.28: The raised ulcerated tumour is about 0.5 cm. thick and 1.5 cm. in diameter with slightly everted edge; surface rough but sligbtly moist and covered by a very small amount of sticky greyish brown matter. Smear preparation from this shows that numerous spirochaetes are present, together with large variety of bacteria.

27.9.28: Under local anaesthetic, $\frac{1}{2}$ grain of cocaine and two drops of adrenaline injected deeply, the tumour was removed by a circular incision and dissected out, to a depth not exceeding 0.5 cm. The raised border of the tumour itself was found to be friable and to bleed easily on manipulation. The wound was closed by a couple of interrupted silk sutures. The wound was not treated and is healing slowly. The goat is now quite healthy and in good condition; it is being kept under observation for a further period to see whether any of the other excoriations will develop. The excised tumour was situated to the left of anus, involving the edge of mucosa. It has roughly the form of a button, with greyish red to brown ulcerating surface about 1.75 cm. in diameter and 0.5 cm. thick. It is covered by a small quantity of smeary exudate and bleeds easily when manipulated. The edges are slightly everted over the stalk, which is covered with skin.

Histological Findings (Specimen No. 8483).

Sections were cut diametrically through the middle of tumour. On both sides the epidermis becomes raised perpendicularly for a short distance as it covers the body of tumour. Its free edge ends abruptly by going over to a loose unarranged tumour parenchyma of squamous epithelium, mixed with a good deal of inflammatory cells. There is marked thickening of the epidermis at the side, as it suddenly merges into the tumour. The thickness of epidermis is increased and appears perforated at intervals by round and oval spaces representing the papillae of corium in section. The corium contains a very large number of round cells. At the edge of the tumour this perforated appearance becomes so pronounced that the epidermis appears to become a reticulum of strands consisting of a layer or two of germinal cells. The intervening spaces are filled by round cells, neutrophiles, and exudate. The transition from the epidermis to the tumour parenchyma is very distinct here. Further towards the centre the continuity becomes lost, but the same arrangement prevails, i.e. chains of basal cells sometimes bent on themselves to form tube-like structures or surrounding small nests of squamous cells. This arrangement alternates with loose masses of epithelial cells of undifferentiated type, which have characters between those of a germinal and a spinous cell, both as regards staining properties and amount of cytoplasm. The parenchyma of tumour with its raised ulcer-like surface is formed thus by a layer 3 or 4 mm. thick, consisting mostly of such ill-defined epithelial cells loosely arranged in strands or nests and profusely intermixed with neutrophiles. At many places haemorrhages have taken place near the surface. The whole structure is supported from a connective tissue bed, with large amounts of round cells, by a very fine connective tissue stroma. In one part the epithelial cells are packed together, resembling squamous cells with horn-pearl formation. Some of the cells are swollen and degenerate like the so-called cancroid parasites. The rest are undifferentiated epithelial cells. Only a few mitotic figures are to be seen here and there. The sebaceous glands of skin close to tumour show a peculiar proliferation of the cells of their basal layer. These chains of deeply staining cells outline the lobulation of the glands distinctly. Faint remnants of sebaceous glands can also be distinguished in the depth of tissue under tumour. Sections stained by silver impregnation method of Levaditi showed several elongated slender black bodies, either solid and thready, or like a string of beads, usually terminated at one end by a lanceolate head or knob. It seems that these represent the spirochaetes which were very numerous in tumour just before excision.

Diagnosis.

Baso- and spino-cellular carcinoma. Mixed form.

Comments.

The development of this tumour was very slow—at one time considerable regression took place. This may explain why the tumour has the characteristics of a cancroid. This may have been brought about by the long-continued irritation caused by injury and excoriation of a pre-existing basal-cell carcinoma. On the other hand, the spinous-cell carcinoma may be primary and the admixture of undifferentiated cell may be due to the fact that the tumour is relatively young. A third possibility is that the tumour is really a basal-cell carcinoma of the keratinizing variety, i.e. carcinoma basocellulare parakeratodes.

It is very difficult to say to which type of epithelial tumour this case belongs. It is for the present, therefore, left as a mixed form of the two types.

GOAT No. 17296.

Clinical Observations.

17.6.27: Angora she-goat, aged. Very poor and miserable condition. The body is extremely emaciated, head hanging and ears limp; dull eyes, and hairy coat soiled and tattered. The animal is being continually chased and butted by the others. The left horn is broken off and the hair around the stump and below it, on face, is matted together with dark, stinking matter, partly dried to crusts. The stump of horn itself is covered by rough, black lumps of the same matter, which, when removed, reveals a cavity opening into frontal sinus. The wound has an intense penetrating foul smell, and is partly grangrenous and filled with fly larvae in various stages of growth. The left nostril discharges a sero-purulent fluid. The left eye is covered by a purulent film and the cornea shows considerable opacity. There are distinct circumscribed, fairly firm swellings palpable in the parotid, intermandibular, throat, neck, and prescapular regions on the left side only. These are painless apparently and vary from the size of a walnut to that of a man's fist, in the case of the prescapular. The perineum is normal. Smear preparations from the slimy matter revealed only a mixed bacterial flora. No spirochaetes are present, but large amounts of black pigment, both free and hoarded in large mononuclear cells, can be distinguished.

18.6.27: Animal lying down, very weak and unable to rise. Pulse and respiration hardly perceptible. Killed for post-mortem examination.

Post-mortem Findings.

Blood-smear negative. Marked cachexia. Partial obstruction (pressure stenosis) of the pharynx, oesophagus, and larynx by the enlarged retropharyngeal, cervical lymphatic glands. Gangrene of the left horn stump and frontal sinus with numerous fly larvae and pigmented tumour formation at edge of skin. Septic metastases in all lymphatic glands of left side of head and neck, as far as and including prescapular. The liver also shows metastases. Purulent panophthalmia more marked on left eye. Catarrhal rhinitis. Greenish pigmentation of kidney medulla. The affected enlarged lymphatic glands on left side were rather soft in consistence, but not fluctuating, on section presented a greyish, moist, somewhat spongy appearance, with very foetid smell, and reddish-grey fluid oozing out freely. Smears from this showed a profuse growth of short coccobacilli sometimes in chains. The respective sizes of these glands were about as follows: parotid, hen's egg; medial and lateral retropharyngeal, same; cranial and caudal cervical, duck's egg; prescapular, man's fist. Those on the right side were unaffected and normal in appearance. An irregular greyish to black ragged growth is found on the skin round the horn stump. It is irregularly lobed, some parts being like small stalked outgrowths and covered by tough black crusts.

Histological Findings (Specimen No. 6985).

Tumour and metastases. A strong connective tissue stroma is arranged in arborescent fashion supporting whorl-like masses of tumour cells between its branches. Following the many depressions and processes of the surface of tumour, a thick layer of necrotic tissue and accumulations of inflammatory exudate and pigment forms the external covering. The contact zone between the two is fairly sharply defined, and is characterized by numerous blood-vessels and capillaries. The tumour tissue below this is remarkably free of any inflammatory cell infiltration. The arrangement of the tumour cells themselves varies somewhat, but the general impression received is similar to that of the other described tumours, i.e. sarcoma-like uniformity of arrangement, with distinct epithelial staining properties of nuclei and protoplasm. Pigment (melanin) is present in large amounts and gives the black colour to tumour. It is produced by the tumour cells, some is hoarded in connective tissue and a good deal is contained in the necrotic outer layer. The tumour cells appear more closely packed together and stain a deep purple colour. In parts they are arranged in definite solid strands, often in slender processes, one or two cells thick, whereas in others the cells seem scattered uniformly in small whorls in between the stroma. These cells have polygonal, spindle, or dendritic shapes, but the nucleus is round or oval and contains one or more well-formed nucleoli. Mitotic figures are very frequent. This is significant when considered with the widespread metastases in the liver and in the whole chain of lymphatic glands on that side of the head and neck. All these glands show the same picture, i.e. infiltrating foci of tumour tissue, identical to that of parent growth. These are surrounded by extended necrobiotic changes of the lymphatic cells. Pigment is present, but to a very limited extent only.

Diagnosis.

Pigmented baso-cellular carcinoma of the base of horn with extensive metastases in lymph glands of corresponding side of head and neck and in the liver.

Comments.

This tumour has certain points of difference from those already described. The great frequency with which mitoses are present in the tumour cells and the extreme malignancy shown might lead one to think that the tumour was different. The structure, however, together with the pigmentation, leaves little doubt that it is an epithelial tumour of the basal cell variety. There is absolutely no sign of keratinization. It would be interesting to know what factor determines the malignancy seen in this case and 17297.

GOAT No. 17297.

Clinical Observations.

17.6.27: Angora she-goat. Aged, condition poor. A large pedunculated black tumour is attached to the left of vulva near its upper commissure. It has only a very short neck and is about 4 cm. in diameter. Under the tail in the depth of the depression, and covered partly by a thick cheesy mass, is a small pink protuberance the size of a pea; it is soft and smooth and bleeds easily when scraped lightly. The larger tumour is covered by hard, dry scabs. When these are removed a dirty greyish-black, purulent fluid is found covering the easily bleeding, spongy black surface. There is a putrid odour from this region and handling seems very painful to animal.

A smear from this material shows a varied growth of yeast cells, cocci, bacteria of all kinds, but no spirochaetes.

23.6.27: Fly larvae are found around tumour, they are busy burrowing their way into it. These maggots were destroyed with turpentine.

30.6.27: Maggots have disappeared altogether from the larger tumour. In the depth of the tail recess two pea-sized pink soft protuberances are seen.

9.7.27: The small pink growths under the tail have decreased in size. Large tumour is covered with thick tenacious crusts of dry exudate, blood, and faeces.

22.7.27: A few maggots have appeared and are burrowing into tumour.

4.8.27: Smear from surface of tumour shows for the first time the presence of spirochaetes in addition to the varied bacteria present.

11.8.27: The tumour is very ragged in appearance as if it had been torn down the middle. There are a few maggots present and the surrounding tissues are much soiled and caked over with black exudate. The vulva is much swollen, especially on the right side, so that it is distorted and pushed over to the left.

15.8.27: Tumour much decreased in size; the centre has been eaten away by maggots, which are still occupying the cavity. The appearance now is that of a cup-shaped, ragged, and pitted or spongy cavity attached to skin by a slightly narrower and shallow base. The right side of vulva is still much swollen and distorted to the left, the swelling being doughy in character, and reddened.

2.9.27: Maggots present in the ulcerated tumour. Right side of vulva stands out swollen and painful. The animal shows great uneasiness, constantly biting, licking, or horning the parts surrounding tumour. It flicks its tail continuously and stamps its feet, and is generally very sensitive to flies settling on and around tumour. It tries to creep and lie down into darkest corners of box, or in between other goats. The condition of animal is naturally very poor, even though eating fairly well.

13.9.27: A new crop of young fly larvae occupies the tumour, which consists now of a thick-swollen and ragged ring, surrounding a much-pitted and widened bed, below the level of skin. All that is left of the original tumour is the hollowed out and widened stalk, in which the maggots have burrowed. The animal shows signs of intense and constant irritation.

23.9.27: No maggots visible. The tumour has now the appearance of a large elevated flat ulcer about 10 cm. in diameter and 3 cm. thick, extending above vulva to anus, and further down to the right of vulva, the latter being pushed over to the left. The surface is black, roughly lobulated, or pitted and fissured and covered by slimy, stinking, dark brown exudate. The actual swelling of vulva has now subsided somewhat.

7.10.27: No maggots present; putrid greenish-grey, necrotic areas over bed of tumour; near the anus a small papillomatous greyish swelling has made its appearance. Smear preparation still shows numerous spirochaetes in addition to the varied bacterial flora.

15.10.27: Tumour still larger, flat, with very irregularly lobulated, gangrenous surface. Numerous maggots present. Animal seems in great distress most of the time. Stamps its feet, flicks tail, scratches with horn, bites and licks anus and tumour almost viciously, rubs itself along wall, lies down in corners, with head to tail, in attempting to evade the flies. Defecation is still unaffected, but during the act short grunts are emitted.

19.10.27: Defecation slightly impaired and painful. Irritation very severe.

25.10.27: Large numbers of maggots eating away tumour Animal feeds fairly well, but very poor and weak.

2.11.27: A good deal of the tumour has been eaten away by the maggots. The surface is pitted and fissured, partly covered by crusts and necrotic tissue. The vulva is still swollen, but much less than before. The tumour measures now only 6 cm. across and 1.5 cm. in thickness. On account of the great suffering due to maggots, these were killed off with xylol. This substance destroys them practically instantaneously and does not seem to cause any additional discomfort to the animal.

3.11.27: The tumour is free of maggots and has contracted considerably, being also much cleaner than it has been for a long time. The goat, however, died during the day on being turned out into paddock.

Post-mortem Findings.

Marked cachexia. Primary tumour between anus and vulva in the form of a shallow ulcer with indurated border. It extends transversely downward and to the right of vulva. Extensive metastases in the supramammary, sublumbar, mediastinal, bronchial, renal, and smaller pelvic lymphatic glands, in the lungs, heart, pancreas, liver, and both kidneys. In the latter the fibrous capsule is distended by an albuminous fluid, probably serum. This is probably due to presence of a large haematoma around one of the metastases on cortex. Acute purulent broncho-pneumonia of left lung. Hydropericardium.

Histological Findings (Specimen No. 7481).

The tumour presents many unequalities, the surface being deeply cut into by fissures. The whole tumour has a somewhat varied appearance. The greater part is made up of a large amount of connective tissue fibres, running in various directions, forming the stroma of large masses of epithelial cells. These are somewhat similar to the basal cells of epidermis. Near the surface there is a vascular zone which is covered by a necrotic layer of exudate. The tumour cells themselves vary much in size and shape. They are large, rounded or oval, in some parts. In others they are elongated and spindle-shaped and thrown into more or less parallel directions when lying near or amongst the fibroblasts forming the stroma. There is some pigment (melanin) in the cells, but only in localized areas. These cells penetrate much deeper than in the other tumours. They lie in some places right amongst the muscle fibres below the subcutis. Mitotic figures are fairly frequent. The numerous metastases in kidney, liver, lymph glands, heart, pancreas, lung, etc., present a most uniform appearance, being composed essentially of round, or irregular, epithelial cells packed fairly closely together and resembling the picture of a round cell sarcoma. The metastatic foci in the various organs are not encapsulated, and in the case of the kidney the remains of atrophied and compressed tubules can still be seen isolated within the tumour tissue. Very little pigment is formed in most of the metastases, those in supramammary glands show pigmentation to a greater extent. Mitoses here are rare as compared to parent tumour.

Diagnosis.

Pigmented basal-cell carcinoma, with metastases in most internal organs and lymph glands.

Comments.

This tumour in appearance and behaviour resembles very much the previous case, i.e. 17296. Here again there was no sign of keratinization.

GOAT No. 17298.

Clinical Observations.

17.6.27: Angora she-goat. Aged, in poor condition on arrival. Skin of face and perineum is pigmented in patches. Skin of buttocks somewhat thickened, harsh, bound, and covered with desquamated epithelial flakes (not due to mange). Vulva much thickened and swollen; on the left side it has a deep wound 2.5 × 1 cm., as if due to laceration with some blunt object some time previous. This wound is covered by tough scabs, which after removal leave a dirty, granulating, fissured bed. This contains foul-smelling greyish pus, and bleeds very easily when handled. There is also a small superficial excoriation of skin about 1 cm. in diameter just above anus. This, however, is clean and shows hardly any inflammation. Smear from exudate of wound shows many bacteria, mostly of short type, a few of the fusiform type, but no spirochaetes.

23.6.27: Dry exudate and blood-crust closely adherent to wound on vulva. No spirochaetes.

4.8.27: Vulva wound appears deeper and is covered by thick blackish crusts as before. Smear from bed of ulcer shows a rich growth of short cocco-bacilli. The small ulceration above anus is somewhat moist, and smears from it show numerous spirochaetes, with hardly any bacteria.

11.8.27: Vulva much swollen; crusts have been removed by force, probably horning, as the ulcer bleeds and is covered by blood-clots.

6.9.27: Left vulva lip swollen. The wound along its centre has widened and deepened. It presents a ragged, ulcerated, and spongy bed. There are granulations at edge of skin, and crusts cover the whole wound or ulcer.

23.9.27: The ulcer remains about the same; there is a slight greyish, purulent discharge from beneath the scabs. On the vulva near the ventral commissure, a small granulomatous, easily bleeding growth has appeared. It is about the size of a hazel-nut.

15.10.27: Animal shows signs of irritation, flicks tail continually, and is sensitive to handling of perineal parts.

19.10.27: The vulva is now much deformed. The left side with ulcer is greatly swollen and the aperture is displaced to the right.

29.10.27: The appearance of perineum has changed completely. Through the swelling of underlying tissue, the bed of the ulcer on left lip of vulva has become raised and everted. It now presents an expanded, mushroom-like appearance with ragged surface, measuring 3.5 cm. high and 2.5 cm. wide. It is covered by a slimy, purulent matter, partly drying up into crusts. (See figure.)

2.11.27: Tumour has somewhat enlarged, measuring now 4 cm. × 2.5 cm., and becomes practically confluent at lower extremity with the small tumour near ventral commissure of vulva. The animal is in poor condition, but feeds well and is still fairly lively.

21.11.27: Tumour about the same, but animal is worried by flies. It lies down in corners with its tail close to the wall, or creeps in amongst other goats.

17.12.27: Animal is much worried and distinctly poorer in condition. It has periodic fits of intense irritation of perineum. It suddenly bites and licks furiously at and around tumour, runs in circles, and jumps in and out of the water-trough and behaves generally as if in great distress.

22.12.27: The tumour on left side of vulva is flat and expanded as before; the surface presents a spongy surface but no maggots can be seen. The supramammary lymphatic glands on palpation show no distinct enlargement, although the left is more prominent than the right.

28.12.27: Animal is standing with head low down in a corner of box, continually flicking its tail and stamping its hind-legs. Now and then it bites or licks round the tumour. It also tries to bite other goats standing near by.

3.1.28: Tumour presents raw, ragged, raised edges on left side of vulva. Supramammary lymphatic glands on left side feel slightly enlarged.

24.1.28: Animal is in very poor condition; bleats very feebly when the very tender affected parts are handled. It suffers still from the marked irritation of tumour even though maggots are not present. It lies down most of the time with head thrown sideways to the perineum. It gets up frequently and wanders about and repeatedly jumps in and out of the water-trough. The supramammary lymph glands are unequal in size, the right one the size of a french bean, the left like a small walnut.

30.1.28: As above, symptoms of continuous and intense irritation. Animal seems to work itself up into a frenzy at times, runs around, bites and scratches itself, even tries to bite other goats and persons standing by. Then it assumes a straining attitude as if trying to defecate but nothing is passed. Defecation is painful and takes place with some difficulty as evidenced by groaning. Vulva is swollen and retracted, the tumour on its left is practically the same.

7.2.28: Animal stands most of the time in a straining attitude, with hocks flexed and back arched. The vulva is much enlarged and swollen, especially the right side, which reaches the size of a hen's egg. No maggots are present.

17.2.28: Marked emaciation, straining attitude most of time. Mouth and lips are soiled with slimy brownish matter from constant biting and licking tumour and the surrounding tissues. The left supramammary gland is the size of a walnut and firm to the feel. The right one about normal size. The tumour consists of a large ulcerated, ragged cavity on the left lip of vulva. The edges are raised and indurated and the rest of vulva is greatly swollen.

25.2.28: Animal crouching down with arched back most of time; the tumour is in a repulsive semi-gangrenous state.

1.3.28: The left supramammary lymphatic gland is now the size of a small orange and firm. The right one is still about the size of a french bean.

2.3.28: Under complete chloral anaesthesia (2 g. intrajugular) the left supramammary gland was removed surgically for transplantation purposes. The enucleated gland measured 4 cm. in diameter and had a rather soft, semi-fluctuating consistence. On section the centre contained a small amount of thick, oily, greyish fluid with foetid smell. The substance around this was rather soft and friable and greyish in colour. Very little glandular tissue was left, forming a shell around the tumour tissue. The whole appears enclosed in a connective tissue capsule. Some of this tissue was used in transplantation experiment No. 7.

9.3.28: Inflammatory oedema at seat of operation. Drainage provided. Temperature of animal rising. Sniffing, nasal catarrh, and inspiratory râles.

11.3.28: Animal much worse, lying down. Respiration difficult.

13.3.28: Found dead in the morning.

Post-mortem Findings.

Marked cachexia. Gangrenous pneumonia. Ulcerated tumour vulva, with two metastatic foci, the size of peas, in remaining right supramammary gland.

HISTOLOGICAL FINDINGS (SPECIMEN NO. 7831).

Strong connective tissue stroma with mixture of smooth muscle. Amongst this are strewn numerous cylinders and strands of epithelial cells of various sizes and shapes, generally with centres of homogeneous, stratified substance (keratin). The tissue consists of numerous slender projections, which are very vascular near surface and are covered by a necrotic layer. There are also a number of superficial haemorrhages. The glandular tissue of supramammary gland is separated from the newgrowth by a fairly thick, loose connective tissue capsule containing much lymphoid cells. The newgrowth consists essentially of spinous epithelial cells, in most parts arranged in solid masses or more usually in compact rings and strands. Many neutrophiles are swollen, degenerated cells (cancroid parasites) are present. In other parts again the epithelial cells grow into irregular strands of elongated cells penetrating the lymphoid tissue like roots. Many show mitotic figures. (See figure.)

DIAGNOSIS.

Spino-cellular carcinoma (cancroid), with metastases in both supramammary lymph glands.

COMMENTS.

This case illustrates very well the intense irritation, amounting to torture, that is suffered by an animal affected with cancer.

GOAT No. 17299.

CLINICAL OBSERVATIONS.

Angora she-goat. Aged, condition poor.

17.6.27: On the upper surface of the left ear, extending from its middle towards the tip, is a large, sessile, black, stinking tumour. It is rounded in outline and flattened, about 6 cm. in diameter. The surface is thrown into multiple lobes by the many fissures and crevices present. These are filled with greyish-black, smeary, foul-smelling substance, mixed with necrotic matter. It is covered by thick, hard, partly dried-out scabs, which, on being removed with some little force, leave the tumour parenchyma exposed and bleeding profusely. The latter consists of a soft, black mass, streaked in places by the presence of greyish connective tissue. The animal appears to suffer considerable pain when ear is handled.

23.6.27: The tumour is much the same, only it is covered with blood-clots and crusts, evidently bleeds frequently when head shaken or ear knocked against objects. Smear preparations show no spirochaetes but numerous bacteria with racquet-shaped spores and cocci.

The dry scabs and the gangrenous matter curetted lightly and tumour generally cleaned. A small piece was then removed for histological examination. The operation was done without anaesthesia, and proved rather painful. The cut surface was extremely vascular and bled profusely, haemorrhage had to be stopped by cautery. The stroma was extremely rich in blood-vessels. The tumour was cleaned with tincture of iodine and a protective suspensory bandage placed on it to prevent unnecessary injury and bleeding. In order to try and get the tumour fairly clean, to facilitate transplantation, the whole affected area was kept in a bandage, regularly cleaned, bathed, and packed with disinfectants. Many disinfectants were tried, long baths with Dakin's solution, magnocid, ichthiol and alcohol, and finally corrosive sublimate in alcohol, strength 1 in 100. The result of all this was that, after two months, the tumour had grown tremendously, more than doubled its size, but was as septic as ever.

9.9.27: The newgrowth now presented a pitch-black cauliflower appearance, the size of a man's fist. It evidently caused considerable irritation judging by the frequent attempts of the animal to scratch it, even through the bandage. The growth is now so heavy that, unless constantly kept in a suspensory bandage, great injury and haemorrhage is caused by animal shaking its head and scratching. The crusts still form over the surface, and from some of the deeper fissures greyish stiff, thin blades of cartilage up to 1.5 cm. in length grow forth. Once or twice, when bandage was not renewed every two days, maggots invaded the tumour under bandage, and seemed to cause greatly increased irritation.

Condition of animal is very poor, in spite of fairly good feeding.

20.9.27: The whole newgrowth bathed in warm magnocid solution for half an hour, and carefully cleaned. As much of the surface as possible was curetted free of gangrenous matter. Examined the regional lymph glands for metastases but none could be seen or felt.

The growth was then removed surgically by a simple linear incision, severing the ear just proximally to the tumour. The skin was first retracted toward the head. The main blood-vessels were ligatured and the edges of skin approximated by a few interrupted sutures. Pieces of this tumour selected, as clean as possible, were cut out, or punched out, with a canula and were transplanted subcutaneously into (*a*) the other ear of the same animal, (*b*) ears of other animals, and (*c*) perineum of other goats. (See transplantation experiment No. 6.)

Complete healing of the left ear stump, with minimum of attention, was rapid and uninterrupted. The right ear with transplant developed a swelling at first, this became fluctuating, and discharged thick, pus-like matter, smears of which were swarming with short cocco-bacilli and chain-like organisms.

19.10.27: Condition of animal distinctly improved. Seat of transplant on right ear practically healed up.

28.11.27: Gaining in condition. Ear stump completely healed up, epithelium again covers edge of ear.

14.1.28: Condition very good, otherwise healthy.

30.11.28: Same, no signs of recurrence or other disturbances, i.e. after 14 months.

Histological Findings (Specimen No. 7385).

A strong connective tissue stroma arises from the subcutaneous tissue of the ear at various points in the form of multiple strands branching and curling round to enclose or support the round masses of tumour tissue. This gives the surface its lobulated appearance.

At the base the tumour practically lies on the conchal cartilage, separated from it only by a thin connective tissue layer. Towards the edge of the tumour the skin seems to have been lifted up and pushed out or distended by the growth under it. The epidermis has a thin stretched appearance. It covers the tumour externally for a short distance only. At intervals long, slender, processes of epithelium from epidermis dip in, deep into the tumour, to join some stroma strand, or to become lost in the parenchyma of tumour. The appearance of the tumour itself is monotonous in its uniformity, and very much like a sarcoma. Melanin is distributed in rather irregular fashion in the tumour cells, some lobules having quite a fair amount in a few cells, while others again have none at all. Extensive hoarding is seen in the stroma at various points, even in the connective tissue outside the tumour itself. This produces the mottled greyish and black colour seen macroscopically. Under higher magnification the tumour cells have a very uniform appearance mostly arranged in a loose fashion, i.e. separated by empty space as if the delicate and sparse cytoplasm of each cell had partly shrunk around the nucleus. Near the edges covered by epithelium, the cells of the tumour take on an elongated spindle shape. The basal cells of epidermis are likewise elongated, at right angles to the basal membrane, and appear as if " shelling out " and merging into the tumour parenchyma. It is at these places in connection with epidermis that pigment formation in the subjacent tumour cells is most marked. Numerous mitoses can be clearly seen, but nowhere is any indication of keratinization visible. One or two tough processes which grew out of the tumour fissures consist of fibrous and cartilaginous tissue. Their significance is not understood, as the pieces were cut out from the living animal, but could not be seen again after removal of tumour.

Diagnosis.

Pigmented baso-cellular carcinoma.

Comments.

This tumour was allowed to grow to a considerable size. In spite of the rapid growth and numerous mitoses present, no metastasis occurred, nor did any of the transplants take. With regard to the latter, it should be said that, owing to cauliflower-like structure, it was practically impossible to obtain any aseptic material from the tumour for transplantation. It seems thus that, although numerous mitoses are usually associated with fast-growing tumours, they are not necessarily the factor concerned in causing malignancy.

GOAT No. 21957.

Clinical Observations.

17.11.28: Angora she-goat. Aged, condition rather poor. Many pigmented maculae on ears, face, udder, perineal skin, and also on body skin generally. Incisor stumps worn very short and covered with tartar. Right eye shows a catarrhal discharge, and the inner two quadrants show a smoky opacity of cornea and a small lentil-sized shallow ulcer. The hair round tail and perineum is soiled with partly dried exudate, blood, faeces, and dirt. On lifting the tail, a large pink, granulating growth is seen filling the whole recess under the tail, so that the anus is only visible as a slit in the middle. To the left the tumour consist of a flat, granulating, pink, moist swelling 1 cm. thick, while on the right it is rounded and bulging, reaching a thickness of 2 cm. The greatest width is 5 cm. from side to side, and 3 cm. from top to bottom. It thus extends between the two tail folds, and reaches practically to the level of the upper vulva commissure. Here it recedes to the level of the skin, but a few shallow ulcers or excoriations are seen on the vulva. (See figure.)

The tumour is covered by slimy, greyish, stringy matter mixed with faeces and dirt. This keeps the tumour moist and gives off a very foetid odour. The animal is very sensitive to handling of the parts, and flicks its tail and stamps its feet when flies settle on or near tail region. It is also fairly tender when the tail is lifted.

The udder is enlarged and lactating so that the supramammary lymp glands cannot be easily palpated. Urination takes place normally. Defecation is fairly painful, but the anus dilates to normal extent, the tumour reaching the edge but not affecting its elasticity. Smear from the surface of tumour reveals mixed forms of bacteria, but no spiral organisms.

20.11.28: A small piece about 2 × 1 cm. was removed from the left edge of the tumour for histological study. Local anaesthesia was complete after deep injection of $\frac{3}{4}$ grain of cocaine and three drops adrenalin. The parenchyma of tumour is firm and somewhat tough to knife, and has a streaky greyish appearance on section. Bleeding from tumour wound was stopped by application of ferric chloride solution, followed by a thin tampon of absorbent cotton wool, as no bandage could be conveniently applied. There was a slight increased swelling on the two following days. Bleeding was negligible and in four or five days the seat of incision could not be distinguished from the rest of tumour.

4.12.28: The condition of animal remains the same, it feeds well and is fairly lively, but resents flies settling near tail region. There are no maggots. Tumour shows no appreciable progress.

HISTOLOGICAL FINDINGS (BIOPSY SPECIMEN NO. 8639).

This tumour presents a fairly uniform appearance. The stroma supports a fan-shaped arrangement of crypts and strands, solid or hollow tubes of epithelial cells. In some places undifferentiated cell types, in others going over to the spinous cell, with horn-pearl formation. The surface is covered by a thin layer of necrotic tissue and exudate cells. Directly under this is a zone consisting of a rich network of small blood-vessels. This tumour presents no other striking characteristics.

DIAGNOSIS.

Baso-cellular and spino-cellular carcinoma (mixed form).

COMMENTS.

On account of the restricted horn-pearl formation and the tubular arrangements of basal cells, this tumour might also be classed as a carcinoma baso-cellular parakeratodes. It falls under the group of ill-defined or mixed form discussed in classification. From the clinical appearance it seems quite probable that the basal cell part was the primary tumour, while the spinous cell form developed later.

HISTOLOGICAL EXAMINATION OF TUMOURS REMOVED SURGICALLY AT VOGELSTRUISLEEGTE, 20.8.27.

GOAT No. 7256 A.

Aged Angora she-goat. Small pedunculate tumour 1 cm. in diameter, non-pigmented, attached superficially to skin at perineum. The tumour consists of a delicately branched connective tissue stroma arising from the short stalk. It gives support to large round masses of rather loose, very uniform tissue, at first glance somewhat resembling a spindle cell sarcoma. At the base, i.e. around the stalk, the epidermis is arranged in normal fashion with sweat and sebaceous glands and numerous blood-vesssels. In places the epidermis is markedly thickened and sends long projections into the subcutis. In others it seems to fork out, sending long thin arms surrounding the above-mentioned round masses of tumour tissue. In many places one can distinctly see the cells of basal layer becoming as it were dislocated, elongated, and merging into the parenchyma of tumour.

This parenchyma is made up of very uniform, loosely arranged cells supported by a fine reticulum or stroma, running in all directions in trellis-like formation. The cells have rather large nuclei, round, oval, or spindle-shaped, with a moderate amount of granular chromatin. There is very little cytoplasm and mitosis is very rare. The stroma varies from place to place, in parts it consists of few fibroblasts with thin strands of fibrous tissue, in others the fibroblasts form thicker strands along which strings of tumour cells can be recognized, resembling the germinal layers of epidermis.

At various places near epidermis small restricted areas are present in which the tumour cells show hoarding of granules of black pigment (melanin). The tumour is only partly covered by a thin layer of epidermis, the rest is exposed and shows a layer of necrosed cells with haemorrhages and profuse exudate. The neutrophiles and lymphocytes infiltrate the parenchyma to variable depths. The blood supply is relatively rich.

Diagnosis.

Baso-cellular carcinoma.

Comments.

This tumour is not quite identical with the solid type of baso-cellular carcinoma described in textbooks. It is, however, closely allied to it and for ordinary purposes can be considered as such. It is a good example of the very lightly pigmented tumours which form a link as it were between the non-pigmented basal-cell carcinoma and the melano-carcinoma.

GOAT No. 7256 B.

Angora she-goat, 4-tooth. The greater part of vulva swollen and hard. It shows a deep ragged ulcer superficially, black in parts and painful. This was removed by a deep incision, but, the swelling and black pigmented parts not being circumscribed, some of the black tissue had to be left behind. The material removed was thus for the greater part vulva tissue with the deep ulcer in about the centre, extending to, but not involving, the vulva mucosa. The pigmentation was general, but very marked under the mucosa of vulva. Under the low power the following features can be noted superficially: a very vascular superficial zone with numerous fairly small blood-vessels. Here and there shreds of epidermis penetrate into the deeper parts, to become lost in single strings or small strands of epithelial cells further down. Beneath this is a very heterogeneous mass of connective tissue, muscle fibre bundles, blood-vessels, and nerves. Much convoluted sweat glands are present, and throughout one sees black granular pigment (melanin) in larger or smaller clumps. On account of this pigment, it is very difficult to distinguish the types of cells present, or even to say definitely in which cells the pigment is contained. Under higher magnification the surface presents a peculiar appearance, as if the cells of the rete malpighi were flattened and separated out. The interstices are occupied by blood and exudate cells; in parts there is loss of substance so that a layer of fibrin and blood exudate forms a sort of crust over the underlying tissues. There is, however, no keratinization of the squamous cells. Below the vascular zone one sees epithelial cells in strands or isolated, some distinctly squamous in type, whereas others, especially along the course of the numerous vessels and capillaries in this region, are difficult to differentiate from endothelial cells. The presence of much exudate, round cells and pigment greatly mars the picture. The melanin either lies free in between the cells (probably artefact) or a few granules lie round the nucleus of the epithelial cells. Large irregular, closely packed masses of pigment are often aggregated together in places, thus completely obstructing the view of the cells concerned. It seems thus that the germinal cells, which actually produce melanin only, show a small amount of finely divided pigment, whereas the large closely packed masses of pigment represent hoarding by the melanophores in the stroma. Judging by the presence of pigment in the perivascular tissue, deep in the intermuscular spaces where no epithelial cells have yet penetrated, it is probable that most connective tissue cells become involved in pigment hoarding.

Diagnosis.

Pigmented baso-cellular carcinoma (endothelioma?).

Comments.

This tumour has the characteristics, clinically at any rate, of the so-called "rodent ulcer." It is, however, deeply melanotic and very vascular. The cellular elements resemble endothelium. One might, therefore, be justified in calling this a type of melano-haemangio-endothelioma. This fact illustrates the difficulty of arriving at a satisfactory diagnosis in many cases.

GOAT No. 7256 C.

Angora she-goat. Aged. Small stalked tumour, blackish appearance, about 1.5 cm. in diameter, situated on perineal skin, above anus. The stalk stump is very short and consists of fibrous tissue and a few bundles of muscle fibres with blood-vessels and here and there well-developed sebaceous glands. On the left the stalk is covered by skin for a very short distance. The epidermis is slightly thicker than normal, the papillary projections well-developed, and in parts the basal layer of cells contains granules of melanin in varying amounts. The basal layer is well defined and lies on the normal corium. A little further on the corium becomes infiltrated with numerous round cells and merges into the tumour tissue proper. In a corresponding manner the basal layer becomes less and less distinct, the malpighian layer becomes thinner, until they finally become lost altogether in the tumour tissue. Towards the end the more compact epidermal cells are in direct contact and seem to merge into the looser cells which form the tumour.

The tumour tissue itself is composed of medium-sized cells with little cytoplasm, and nuclei either spindle-shaped, oval, or round, but with about the same chromatin contents and arrangement as the basal cells of epidermis. These cells are supported by a network of fibroblasts and fibrous tissue. They are not packed together, but are separated from each other. In some cases, however, they seem to lie in rows on the stroma. The cells are further characterized by containing clumps of granular pigment (melanin). The pigment is distributed throughout the tumour, but some cells contain much more than others. The stroma is well provided with blood-vessels, especially near the surface. The parenchyma is infiltrated throughout with inflammatory exudate, the surface being also covered by a necrotic layer and small haemorrhages. The right side of stalk, instead of the skin as on other side, shows an isolated stretch of tissue very similar to a squamous-cell carcinoma. It consists of solid strands of squamous cells. These are separated by stroma rich in blood-vessels and with numerous small round cells. There are a number of swollen and vacuolated cells, with so-called inclusions, and ill-defined pearl-nest formation. Although this portion is continuous with the tumour, it is entirely different in structure and, furthermore, contains no melanin. This portion forms only a small fraction of the whole tumour. It is noteworthy that the appearance of the tumour is exactly similar to that of 7256 A, except for the presence of larger amounts of melanin.

DIAGNOSIS.

Pigmented baso-cellular carcinoma, combined with a non-pigmented focus resembling a squamous-cell carcinoma.

COMMENTS.

It would be interesting to know whether this focus originated independently, or whether it is a transitory stage developing from the pigmented tumour. The absence of pigment from this focus and its very circumscribed nature seem to indicate independent development, from a different epidermal layer. One might think of the primary basal-cell carcinoma acting as an irritant and stimulating the adjoining epidermis to true cancroid formation.

GOAT No. 7256 D.

Angora she-goat. Full-mouth. Small pedunculated tumour, less than 1 cm. in size, situated on skin under tail, just above anus. On section greyish and rather soft, covered by dry necrotic crusts.

This tumour is somewhat similar in appearance to 7256 A with certain differences. In the first place there is no epidermis at any point by which an indication could be obtained as to origin of cells. There is less connective tissue stroma. This appears to be younger, the tumour cells are larger and more closely packed together. The cells have the typical epithelial nucleus, rather poor in chromatin, round or oval in shape, with prominent nucleolus, frequently two or even three. There is very little cytoplasm. Melanin is seen in small granules in a few cells only. No mitosis can be seen. Blood-vessels are fairly numerous. The arrangement of the tumour cells has nothing characteristic, it reminds one somewhat of a large, round-cell sarcoma. In places, however, the cells tend to aggregate into nests, and the ones next to the stroma are more or less in string formation. Numerous neutrophiles and round cells infiltrate the whole tissue, but are particularly numerous near the surface, where a fibrinous, partly necrotic layer forms the external covering.

Diagnosis.

Baso-cellular carcinoma. Slightly pigmented.

Comments.

This is probably a very early stage in the development of this tumour. Unfortunately, the adjoining skin from which more information might have been obtained, was left behind in the process of removal.

GOAT No. 7256 E.

Angora she-goat. 6-tooth, very poor condition. Tumour situated superficially on nape of neck (skin) just to the right of median line, size 6 × 3 × 3 cm., elongated and rounded, covered by stinking and moist, sticky exudate. The attendant states that this tumour originated as a simple " wart " of the skin. From injury by scratching the wart started bleeding, and ultimately developed to its present state. The tumour certainly causes irritation. The animal frequently scratches it with its hind-leg; the maggots present are probably responsible for part of the irritation. The tumour is hard and greyish in section with fine convoluted striations. Arising from the base of stalk, a thick connective tissue stroma branches out in all directions in tree-like formation. On these branches and twigs of stroma are arranged chains or strings of rather large epithelial cells, in layers either single, or several cells thick, resting on a basement membrane. These cells have a round or oval nucleus, poor in chromatin; cytoplasm is rather abundant and stains with hemalum and eosine, the purplish mauve characteristic of epithelium. The outline of the cells is either polyhedral or flattened. The arrangement of these strings of cells gives rise to crypt-like or even gland-like formation, the lumina of which are filled with degenerated cells of the same type. Little or no keratinization has taken place, but some swollen cells containing large vacuoles and inclusion bodies are frequent. These cells closely resemble prickle cells of epidermis, but their cytoplasm stains deeper, and the prickles are not distinct. They appear to be a stage in between the basal cell proper and the prickle cells. They certainly show no tendency to keratinization or pearl-nest formation. The covering epidermis may be traced, from the base of stalk, for a little distance over the tumour; here it becomes lost in the ulcerated part of the tumour. At first it is of normal thickness with sweat and sebaceous glands beneath it. Then it thickens (acanthosis), the interpapillary projections become longer and dip deeper into the subjacent tissues. In several places it is possible to note a swelling and loosening of germinal layer of the epidermis. Such cells are similar in appearance to those forming the tumour (see Fig. 36), and in others the continuity of the altered epidermis with the strings of tumour cells appears well established. Apart from the short distance covered by epithelium the rest of the tumour is covered by a necrotic layer of inflammatory exudate, blood, and dirt. The inflammatory cells of course infiltrate the tumour practically throughout, especially in the deep crypt-like cavities mentioned above.

Diagnosis.

Carcinoma baso-cellulare (adenoid type).

GOAT No. 7256 F.

Angora she-goat. Aged. Superficial tumour of skin situated just under tail, above anus, and measures about 6 × 4 cm. It is elongated, cauliflower-like, stinking, partly necrotic, hollowed out, and full of maggots. It is attached to skin by a short stalk 2 cm. across The tumour is covered by thick crusts and on section has a greyish colour and is somewhat hard to cut. A strong connective tissue stroma arising from the stalk of the tumour branches out in irregular fashion to support solid elongated or rounded masses of squamous epithelium which form the tumour parenchyma. Frequently the larger masses show in their centres the typical hornified, lighter red staining cells, or pearl-nests. They are arranged in concentric fashion and are surrounded by more or less flattened squamous cells. The small amount of epidermis which covers the stalk on one side and extends over the tumour for a short distance presents an interesting aspect. In the first place, it is much thickened, with many blunt projections dipping into stroma. The epithelium is entirely unpigmented. In places the thickened epidermis appears to shed cells which are in parts more or less loosely arranged, or in strings of single cells (compare this with 7256 E). The tumour cells themselves show frequent mitoses and most of them also have 2, 3, or 4 nucleoli; keratinization is distinct. The " horn-pearls " are composed of typical faint staining keratinized substance with clumps of granular matter (eleidin). There are large swollen cells often containing dark staining bodies and neutrophiles, or showing several nuclei. Occasionally one sees a giant cell at the edge of one of the pearl-nests. The superficial part of tumour shows extensive haemorrhages with inflammatory exudates pervading its substance to varying depths. The surface is covered by a necrotic layer of exudate, blood, and cell debris.

Diagnosis.

Carcinoma spino-cellulare (cancroid).

Comments.

Although this tumour has so many characteristics typical of the cancroid, there are certain points which give one the impression that the tumour elements are derived to some extent from the epidermal basal cells. It is, therefore, not a straightforward cancroid, but has certain tendencies towards the mixed forms which have already been mentioned.

GOAT No. 14770.

6.9.26: Angora she-goat. Full-mouth, rather poor condition. Hair round anus soiled and matted together by dark, brownish-black, foul-smelling, smeary material. On lifting the tail a large tumour occupies the recess under the tail and extends down, covering anus and upper half of vulva. The tumour is roughly quadrangular, measuring 6 cm. in length by 4 cm. across and 1.75 cm. thick. It is attached by a short stalk to the skin over an elongated area above and to the left of anus. The tumour is very irregular and lobulated in outline, somewhat resembling unhealthy granulation tissue. Its surface is covered by above dark, smeary, sticky material. The central part shows a depression and is of a lighter colour than the periphery. The colour varies from dark reddish brown, at outer edge, to greyish-green brown at centre. The lower part of tumour is covered by a sticky, pseudo-membrane or crust with much dirt and pieces of straw adherent. On pulling this crust off, bleeding is fairly profuse. The whole mass is movable over underlying tissue and appears attached to skin and subcutis. The skin of perineum is not pigmented and shows practically no inflammation. The anus and vulva are patent, defecation and urination taking place with little difficulty.

22.4.27: Tumour has decreased in size considerably, now measuring only 4 × 3 × 2 cm. It is attached by a fairly slender stalk which is now easily visible. It is about 1 cm. above and to the left of anus. The stalk is somewhat flattened and less than 1 cm. thick. The surface of tumour is covered by a slimy, sticky, stinking material; it is greyish green to brown, due to partial gangrene. Smears made from scrapings of surface of tumour show numerous spirochaetes and a varied bacterial flora, with long threads and fusiform organisms. Blood-smear is negative. The lower aspect of the tumour presents a ragged cavity, in which numerous fly larvae are lodged. Hair around breech is matted, soiled by stinking, putrid exudate from perineum and tumour.

27.4.27: The whole tumour has disappeared, also all fly larvae. All that remains is a small oval, raised, ragged area, the stump of the tumour stalk. This shows hardly any inflammation and granulates under the scab. The whole perineum is much cleaner than it has been till now, the smeary material having disappeared with tumour.

2.5.27: Small scab present under tail stump has healed up completely.

This animal has been kept under observation from time to time and is alive to-day and in perfect condition. Note that at no time was any interference attempted either with tumour or afterwards. The fly larvae burrowed right down to the rather slender stalk of tumour, from which the hollow shell of the tumour then fell off, thus ridding the animal of both the tumour as well as the maggots as effectively as if a surgical operation had been performed. The small wound thus remaining healed up rapidly under a scab, and no signs of recurrence have been noted during the 18 months elapsed since. The skin of perineum has remained throughout quite clean, soft, and pliable. The goat is still being kept under observation.

This confirms the fact already noted histologically that these tumours in their early stages are non-infiltrative and very well circumscribed. This applies even more to the stalked variety. This fact undoubtedly explains why it was possible for the fly larvae to eat away the whole of the tumour tissue.

GOAT No. 17292.

Angora kapater. Full-mouth, very poor condition. This animal died on arrival, and through an oversight only a cursory post-mortem examination was made, and the carcase destroyed without collecting material for histological study.

Post-mortem Findings.

Marked cachexia. Chronic adhesive pleuritis with multiple caseous abscesses (Preisz nocard), caseous lymphadenitis of bronchial and mediastinal lymphatic glands. Acute broncho-pneumonia of both lungs with multiple caseous abscesses. Hydropericardium. Left horn was broken off. Stump and surrounding tissues were covered by thick, black, dry scabs, which on removal exposed a cavity leading down to frontal sinus. This contained a mass of gangrenous blackish tissue with slimy, greyish-black, foul-smelling, fluid. The edges of cavity around the base of horn presented a firm, ragged, granulomatous appearance covered by gangrenous material. No metastases in regional lymphatic glands were recorded. This description agrees so closely to that of 17296 that there can be little doubt that the condition was similar except for the metastases in regional lymphatic glands.

GOAT No. 17300.

Angora she-goat. Two-tooth, poor condition and stunted. Arrival 15.6.27.

Over the middle and about 2 cm. above the brim of the left supra-orbital process, there is a papillomatous growth about 2 cm. in diameter. It is sessile and the spike-like projections are hornified. The interstices between these, however, and the base of growth show a small amount of thick greyish purulent matter. The growth is apparently non-painful.

23.6.27: There are signs of slight haemorrhage on growth, probably due to traumatic injury. Half the tumour was removed for histological purposes. The wound painted with tincture of iodine.

30.6.27: The wound as well as rest of growth are covered by thick dried exudate scab, which on removal leaves granulating wound with purulent discharge. Disinfected and cleaned.

9.7.29: Growth decreased in size, tendency to heal up.

22.7.27: Small scab remains; on removal of this, slight bleeding, but no trace of tumour.

2.8.27: Healed up completely, leaving only a small scar behind.

29.9.27: Painted with coal-tar a circular area extending to base of left horn, and including seat of this wart. This area, about 6 cm. in diameter, was painted at intervals (see Experiment No. 10) after scraping off the previous tar crusts.

30.11.28: This painting has now been continued for over a year, but no change can be noted on the painted area, except the loss of hair and a slight thickening and roughening of skin. This animal was selected for tarring on account of its origin from affected flock and because it showed tendency towards wart formation, which may or may not be an indication of predisposition to tumours. The experiment is proceeding.

Part of the papillomatous growth removed surgically from skin above left eye was examined.

Histologically the growth consists of connective tissue, largely infiltrated in patches by round cells and neutrophiles, and sends out multiple finger-like projections which are covered by a rather thin hornifying layer of epidermis.

Numerous forking slender projections of epidermis dip down into the corium. The basal layer is, however, continuous and sharply defined. A few chromatophores are present in corium, and a few only of the basal cells contain a little pigment. The horny superficial layer is fairly thick.

Diagnosis.

Papilloma, with slight inflammatory changes at base, probably due to injury.

GOAT No. 17301.

Angora she-goat. Two-tooth, rather poor condition and stunted. Arrived 15.6.27.

Near inner canthus of left eye a small greyish, papillomatous growth 1 cm. in diameter, is observed. It is sessile, dry, and horny, shedding scales of epithelium. The perineal skin is brownish in colour, but shows nothing abnormal.

30.6.27: The dry, brittle horny superficial projections of wart can be easily and painlessly crumbled away by scraping lightly; this leaves a small hard, raised area, the size of a pea, surrounded by a zone of hairless skin. This thickened piece of skin gradually returned to normal by a process of scaling off, until later the hair grew again.

8.8.27: Animal is healthy to all outward appearances and is in fair condition.

22.10.27: This goat now selected for tarring experiment. Tar applied alternately and in regular rotation in four different places. Areas about 10 cm. in diameter were clipped and tarred thus: (1) on the nape of neck, (2) on the sacral region, (3) left thoracic region, (4) right thoracic region. (See Experiment No. 10.) Tarring was discontinued on the 1.3.28.

3.4.28: Hair is growing well over areas now clean from tar. The skin here shows no marked difference from that untarred, except for a slight roughness.

21.6.28: Quite normal in appearance and condition.

30.11.28: Ditto.

Nothing further was done to this animal. The skin near canthus of eye is normal and has shown no further development. The tarring over alternate patches of body for four months was undertaken with the object of noting whether the slow tar absorption through the skin over fairly large areas would favourize the growth of warts in an animal already with this tendency. The results are so far negative.

This wart-like growth at the beginning may be taken as an example of what farmers think gives rise to a malignant tumour, following on injury, scratching, etc.

On account of the rapid disappearance of the wart, no material was available for histological examination.

GOAT No. 9983.

Clinical Observations.

Angora she-goat. Aged, condition fair.

30.4.27: Hair very long, at the buttocks tangled and matted together by dark slimy material, smelling strongly of decomposing urine, and consisting mostly of faeces, dirt, secretion, etc. On lifting the tail, a fair amount of greyish, sour-smelling, cheesy material is found partly caked under tail. On scraping this off, a small stalked, rounded protuberance, the size of a pea, is found adhering to skin above anus. It is soft, apparently covered by a delicate membrane. Scrapings from the surface of this revealed a mass of bacteria of mixed variety.

11.5.27: Vulva reddened superficially and swollen as if injured mechanically (horning?). The small protuberance mentioned above is partly torn off, hanging on by a shred of tissue, and showing evidences of bleeding. The stump was cleaned, and some material obtained from tumour of goat 14771 was rubbed well into the lacerated stump.

13.5.27: Mass of cheesy matter present under tail. The stump of torn protuberance bleeds easily on manipulation, and is covered by greyish, sticky matter, a smear of which showed a rich and varied bacterial flora, with numerous fusiform bacteria, and short spiral organisms. Stump reinfected by swabbing from tumour of goat 14771.

16.5.27: There is now a small sessile granulating elevation, the size of a pea, which bleeds easily, surrounded and covered by a moist, yellowish-grey sticky material. This consists of a mass of bacteria growing in the serous discharge from granulation, together with decomposing pus cells and skin gland secretions.

18.5.27: Fairly dry and clean; depression under tail shows cheesy material partially covering a small pink swelling, the size of a split-pea. Smear made shows mixed bacterial flora, with fusiforms, racket shapes, and short spiral organisms.

23.5.27: Depression under tail fairly dry, small swelling decreasing in size.

4.6.27: The long, bushy hair around perineal region soiled and impregnated with urine. Swelling has practically disappeared.

13.6.27: Healed up apparently.

6.7.27: No change. Injected subcutaneously, into perineum above anus, swab suspension from goat 14771. Tied an absorbent pad over perineum to keep exudate and urine into continuous moist contact with perineum and thus cause maceration of skin.

11.7.27: Purulent greyish matter discharging from seat of injection. This is kept in contact with skin by above pad.

15.7.27: Stinking, greyish-brown discharge kept plastered over swollen wound, and granulating bed, by absorbent pad.

22.7.27: Wound has cleaned up, and is healing in spite of pad and decomposing matter.

2.8.27: Slightly moist exudate covers wound, which is healing well.

8.8.27: Matted hair and absorbent pad keep moist conditions, wound has healed up completely. On scraping cheesy matter under tail a small pink protuberance found in the same place as the previous one.

26.8.27: Fairly dry cheesy matter present, protuberance bleeds easily when cleaned of adherent matter.

6.9.27: Moist and sticky matter under tail, protuberance appears somewhat swollen.

13.9.27: Same, slight purulo-catarrhal discharge from vulva.

15.9.27: Shorn, condition of animal good. Face and ears have a clear skin, i.e. show no pigmented spots. Recess under tail still carries a small granulomatous protuberance, partly covered by caked cheesy matter.

20.9.27: It was decided to apply tar regularly at intervals to skin of perineum. This animal was chosen and included in the experiment because it already had a growth of sorts on the anal skin, and that tar application might hasten the development of that into some definite neoplasm. The whole perineum was painted. (For tar used, etc., see Experiment No. 10.)

29.10.27: Protuberance under tail still present, but shows no change although included in the tar-painted area. It becomes surrounded by a putty-like black mass consisting of tar and abovementioned cheesy substance.

14.11.27: Perineal skin shows a blackish-brown tinge from tar used. The partly dried crust of tar is scraped or peeled off from skin before each application.

17.12.27: Skin at edge of perineum is slightly thickened and covered by tar crusts. The skin of perineum itself is still soft and pliable. Protuberance present, but shows no progress.

24.1.28: A small cornu cutaneum, about 3 cm. long, has been growing on inside of right ear for some time. In the recess under tail the small tumour is still present; it is now flattened and papillomatous in nature and about 1 cm. in diameter.

3.4.28: The pink granulomatous growth under tail has increased in size, it has a flat, expanded papillomatous appearance and about 2 cm. in diameter, and is generally surrounded by a black sticky mass of tar and detritus. It bleeds easily when scraped even gently.

21.6.28: Still about the same.

30.8.28: Flat papillomatous growth still ±2 cm. in diameter, bleeds on manipulation. Surrounding skin shows no change.

11.9.28: In order to increase toxic effect of tar by increasing absorption, fairly large areas of skin, 15 cm. in diameter, were clipped free of hair, on both thoracic walls and gluteal regions. These were painted with tar on alternate dates in addition to tarring of perineum.

24.9.28: Animal is losing condition and appears sickly, listless, shuffling, stiff gait, lachrymation of one eye.

28.9.28: Discontinued tar application, animal losing condition and not feeding, miserable appearance.

2.10.28: Died during the night.

Post-mortem Findings.

Caries of middle incisors with gangrene of gums. Swelling of liver, with greyish-pink reticulated appearance, due to prominence of interlobular tissue. Much enlarged and thickened gall-bladder filled with fluid yellow bile. Multiple pin's head sized cysts in cortex of kidney. Numerous small hairballs in abomasum, ranging in size from that of a pea to that of a large marble. Small flat papillomatous tumour in recess above anus. Catarrhal enteritis. Tar poisoning?

Histological Findings (Specimen No. 8507).

Congenital fibrosis of liver with extensive proliferation of bile ducts. Cystic kidneys. Tumour above anus presents roughly the shape of a mushroom, i.e. it has a short but transversely elongated stalk about 0.3 cm. thick, supporting a thin flattened, expanded top about 1.0 × 2.0 cm. The surrounding skin comprises a rather thin epidermis, which is continued up the stalk and on lower aspect of expanded part of tumour. There are numerous sebaceous glands which present a peculiar appearance. They are much lobulated and consist of a greater amount of cellular tissue than normal, so that only a small number of fat-containing cells are present in centre. One gets the impression that the basal layer of the gland is thrown into convolutions and that the basal cells are greatly proliferated. The sweat glands are few in number and unchanged. There are numerous scattered round cells in the subcutis.

The superficial expanded portion of tumour presents an irregular surface. The epidermis is continuous at the edges with that of skin, but becomes intermittently greatly thickened on surface. It sends thick processes dipping down into underlying tissues. These are separated by fissures or cavities usually filled with keratin, forming thus a very irregular surface. The epidermis, moreover, is not sharply defined, but blends or becomes continuous at various points with masses of epithelial cells below, which appear to be altered remnants of sebaceous glands. These contain very little fat-carrying cytoplasm, but the lobulated arrangement, and more or less sharply defined deeper convoluted layer of germinal cells, remind one of sebaceous gland structure. Here the proliferation of cellular elements is even greater than under the skin a short distance away. These basal cells, from being more or less in single layers in the depths, become loosened and scattered. They are round, flat, or spinous in character, and merge into the dipping processes of altered epidermis. In places blood capillaries become fairly frequent and even small haemorrhages are present in the superficial layer. There are suggestions of small foci of horn-pearl formation, i.e. swollen squamous cells with disintegrating nuclear substance. Nowhere, however, does one find the uniform loose epithelial cell formation, breaking away from a definite basal membrane as in other tumours of the basal cell type, nor the typical pearl formation seen in cancroids. Although not typical, one might probably call this a type of sebaceous gland adenoma, accompanied by fairly extensive alteration of the epidermis. It is not difficult to imagine a step further in the development of this tumour, in which the basal cells of the gland become more and more emancipated and likewise the epidermis tends to cancroid formation which would ultimately give rise to the mixed form of basal and spino-cellular types of carcinoma which have already been described.

DIAGNOSIS.

Proliferation of sebaceous gland basal epithelium (adenoma?) with accompanying acanthosis. Probably an early stage in the combined form of baso- and spino-cellular carcinoma.

GOAT No. 7256 G.

Angora she-goat. Six-tooth. Ventral vulvar commissure and clitoris swollen, red and encoriated, covered with exudate. This whole region was excised, as it was thought this might be the early stage of a tumour. The epidermis at its junction with the vulva mucosa is necrotic over a small area, so that no epidermal cells are left intact. The bed of this ulcer is shallow and the layer of fibrin, exudate, and necrotic cell debris rests on a mass of connective tissue very heavily infiltrated with round cells, neutrophiles, and other cells. The rest of epidermis and mucosa are fairly thickened, but show no abnormality. The former shows the usual skin glands. The whole corium, however, together with the subcutis, is extensively infiltrated with neutrophiles, these in parts filling small cavities (abscesses) and greatly marring the rest of structure. There are round cell accumulations and numerous young fibroblasts and vascular tissue. In parts, it seems that epithelial cells can be distinguished, but these may be endothelial cells actively dividing.

DIAGNOSIS.

Ulcerative dermatitis of vulva. Whether or not this is a beginning tumour of the basal cell, or rodent ulcer type, is difficult to say, as no definite indication could be obtained from this material.

GOAT No. 14505.

Angora she-goat. Aged, in good condition.

30.4.27: A fresh, bleeding, deep lacerated wound, involving vulva, was discovered, and probably was due to horning by another goat.

There was considerable swelling of parts with purulent discharge. On account of constant soiling with urine, no healing took place. The animal was kept under observation to see whether such a chronic inflammatory process would give rise to neoplastic growth. Spirochaetes and bacteria were found in the wound on various occasions when smears were made. The wound remained in more or less the same state for months; the inflammatory swelling gradually subsided, leaving a deep slit obliquely across the vulva. (See Figs. 42 and 43.)

30.11.28: After 19 months the wound has not yet closed. This slit on the right lip is fairly deep, has smooth sides, and opens up into the vulva.

Slight superficial excoriations with attendant swelling can be seen at intervals near the lacerated part. There is also a slight gangrenous tendency in the depth of the slit. There is still the possibility, therefore, that this may ultimately develop into a neoplasm.

The animal is being kept under observation.

GOAT No. 15690.

Angora she-goat. Aged, in fair condition. This animal was included in experiments Nos. 2, 5, and 10, which gave negative results. It was noticed to have small epidermal protuberances or fringes under the root of the tail as shown in photograph. (See figure.) On these, frequently, faeces, dirt, and smegma-like sebaceous secretion tend to accumulate and, by the to and fro rubbing action of the tail, form small balls suspended by a slender stalk. It was thought that this might have some irritating effect. One of these protuberances was excised and examined, but was found to consist of a fold of skin only, i.e. normal epidermis, with the usual skin glands. The covering epidermis showed no abnormal thickening, keratinization, or other changes characteristic of papillomatous or other growths.

VIII. SUMMARY OF EXPERIMENTS.

Experiment No. 1.—Contact.

One she-goat, No. 11969, was kept in contact with affected animals Nos. 14770, 14771, and 14772 for at least six months. During this period she gave birth to one kid. There was ample opportunity for infection, as the box in which the animals were kept continuously at the time was not large. Later on, all affected animals and available experimental animals were kept together in a small paddock. Apart from No. 9983, which already had a small nodule at perineum, no cases developed amongst the in-contact goats.

Experiment No. 2.—Transmission.

This experiment was carried out by Mr. Bisschop, of this Division. Goat No. 14773 was killed for this purpose and material from the anal tumour used in the various ways indicated below:—

She-goat No. 15689, aged. A small piece of the tumour was implanted into anal sphincter on either side.

She-goat No. 15690, aged. Triturated tumour tissue was injected subcutaneously and intradermally at perineum.

She-goat No. 15691, aged. Piece of tumour was rubbed well into the cleaned and scarified skin near anus.

She-goat No. 15692, aged. The unscarified skin treated in the same way as that of No. 15691.

The results from these four cases were entirely negative.

EXPERIMENT No. 3.

Material obtained by means of swabs from the surface of tumour No. 14771 was used as follows:—

Rubbed into intact mucosa of vulva and anus of she-goat No. 15693.
Rubbed into scarified perineal skin of she-goat No. 15694.
Rubbed into scarified mucosa of rectum of No. 15696.
Injected subcutaneously at perineum No. 15697.
Injected suspension mixed with liquid paraffin subcutaneously at perineum No. 15698.

Results here were likewise entirely negative.

EXPERIMENT No. 4.

Suspension of swab material from surface of tumour No. 14771 was injected subcutaneously near anus of one rabbit and two guinea-pigs. The rabbit developed a tense bluish swelling on the sixth day. It was killed on the eighth day on account of dull state and very large gangrenous swelling near perineum. The lesions resemble those due to *B. necrophorus*. Cultures and smears confirm this organism as cause of the lesion. The guinea-pigs only developed small swellings, which, however, after some time disappeared. The bacillus of necrosis is thus present on these goat tumours.

EXPERIMENT No. 5.

A small piece of the ear tumour was excised from No. 17299 on the 23.6.27. Ground-up suspension of this was injected subcutaneously into the ear of No. 15690 and the perineum of No. 15691. Some was rubbed into the scarified ear skin of No. 14771, suffering at the time from an anal cancer.

Negative results from all three animals.

EXPERIMENT No. 6.

The whole ear tumour, No. 17299, was removed surgically on the 30.9.27. Small pieces, taken as clean as possible, but obviously not aseptic, were implanted as follows:—

(*a*) Into the subcutis of the other ear.
(*b*) Into the subcutis ear of No. 14505.
(*c*) Into the subcutis of ear and perineum of No. 15698.
(*d*) Into subcutis ear of No. 15697. This animal had previously received injection of Indian ink to " blockade " its reticulo-endothelial system.
(*e*) Into subcutis of ear No. 15691.

Result.—In the case of No. 15697, an indurated swelling developed at the seat of the transplant. This reached the size of a french bean, but later softened and discharged a thick greyish matter. The small wound then completely healed up. In all the other cases the implanted material seemed to disintegrate and become discharged.

EXPERIMENT No. 7.

One of the markedly enlarged supramammary lymphatic glands of No. 17298 was removed surgically on the 2.3.28. It was hoped in this way to obtain practically aseptic, metastatic tumour tissue for transplantation purposes. This material, however, proved to be already septic, so that no intrajugular injections could be made. The ground-up tumour tissue was injected subcutaneously into the following animals:—

Female No. 18783 received 4 c.c. into the thigh.
Female No. 18784 received 4 c.c. into the thigh.
Female No. 18785 received 2 c.c. into the right ear.
Female No. 18786 received 2 c.c. into the right ear.

All four cases behaved in the same way, i.e. a small firm swelling developed at the site of the injection and reached the size of a hazelnut in about 10 days. This gradually became soft, and later burst, discharging a small amount of thick, greyish matter. The small wound then healed up completely. No material for section was taken at any time in the fear of disturbing any growth taking place. Results are thus negative.

EXPERIMENT No. 8.

Infection of existing chronic wound of vulva No. 14505 with swab material taken from surface of tumours Nos. 14771 and 17293. Although this material contained large numbers of spirochaetes and bacteria, which developed to a certain extent in the wound, no progress was noted. The wound remained in the same state, i.e. refusing to heal up. It seems doubtful whether micro-organisms have any direct influence on the production of these tumours, unless such influence becomes manifest only after very long periods of time.

EXPERIMENT No. 9.

Large doses of potassium iodide daily, either alone or in combination with arsenic, were tried on Nos. 14771 and 17293. There was a slight reduction in the size of the tumour. This, of course, may be due to spontaneous regression, which has occasionally been noted in such tumours. Since no encouraging results were obtained, the use of this drug was discontinued.

EXPERIMENT No 10.—TAR APPLICATION.

The extensive work done on the artificial production of skin cancer, and the comparative ease with which this is brought about by the application of coal-tar, led to the following experiment. It was reasonable to think that the Angora goat, being so susceptible to spontaneous skin cancer, might readily respond to the action of coal-tar:—

Eight goats were selected; some originated from affected flocks, others from totally different sources. These were painted with tar every three or four days, i.e. twice a week. Six young rabbits were selected and used as controls, i.e. their ears were painted on the inner surface, with the same tar on the same dates. The tar used was an ordinary grade of imported coal-tar, manufacture unknown. The routine procedure of application consisted of clipping the hair, where present, over area to be treated. Before each application the partly dried crusts from the previous treatment were carefully removed by peeling or scraping off. In the goats whose ears were painted, shields had to be fastened to the horns, to protect the eye from contact with the tarred ear.

Particulars of the animals treated are as follows:—

17295, she-goat, full-mouth. From affected flock, skin shows pigmented patches. Painted the whole of perineal skin from 20.9.27 to 7.11.27. The animal then suddenly lost condition and had a severe diarrhoea. It died on the 15.11.27 and showed a marked enteritis, evidently due to some intercurrent infection, or possibly to tar-poisoning. The skin in those parts painted with tar showed no change whatever.

16704, she-goat, aged. Non-pigmented skin. Available animal. Perineum painted since 20.9.27 and still proceeding now after 15 months. No changes can be noted in the treated skin.

11969, she-goat, aged. Pigmented skin. Tarred the inner surface of right ear from 7.11.27 to 11.9.28. From this date tarred outer and inner surfaces alternately. Skin shows a very slight thickening.

15689, she-goat, full-mouth. Non-pigmented skin. Tarred perineum since 20.9.27 and still proceeding now after 15 months. Four areas roughly 15 cm. in diameter were clipped on the thoracic walls and gluteal regions. These were tarred in rotation, one on each date. The animal lost condition rapidly after a month of this. Tarring of the body was, therefore, discontinued. Only the perineum is painted now. The skin of perineum shows hardly any change. There is a slight thickening, but no enlargement of follicles.

15690, she-goat, 6-tooth. Pigmented patches on skin. Tarred upper surface of ear since 20.9.27. In this area was included a chronic swelling and ragged wound due to an ear-tag having torn off. Tarring is proceeding, and since 11.9.28 both ears on both surfaces are tarred alternately. A small piece of skin in tarred area was removed and examined microscopically. Apart from signs of chronic inflammation, no change could be detected. The horny layer was not increased. Four areas roughly 15 cm. in diameter, on thorax and gluteal region, were clipped free of hair and subjected to tarring in rotation, at the same time as the ear.

15696, she-goat, 6-tooth. Pigmented patches on skin. Tarred upper surface of right ear since 20.9.27. From 11.9.28 both the surfaces are painted alternately. So far no change is visible in the treated skin.

17300, she-goat, 2-tooth. Unpigmented skin. Tarred an area of skin on forehead, including the base of left horn, and extending down over the seat of a wart which has since disappeared. Application since 20.9.27, and still proceeding after 15 months. No change in skin can be noted.

17301, she-goat, 2-tooth. Clipped and tarred four areas on body, alternately and in regular rotation. These areas were situated on the neck, rump, and on either thoracic wall. Tarring was discontinued after four months as the animal became very poor in condition.

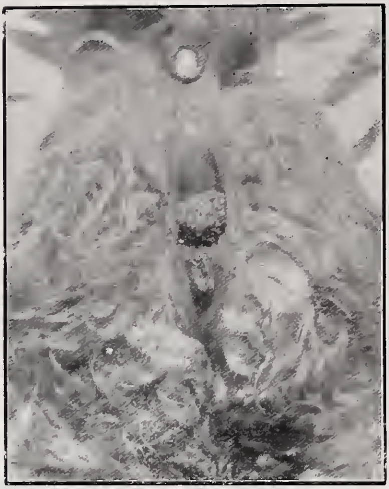
The control rabbits were treated in the same way from the 20.9.27. One died a month after, apparently from tar-poisoning. The five remaining animals all started showing small bleeding nodules on the ears from the third month. After six months, well-developed horny, papillomatous growths were present on all areas. (See Figs. 46 to 50.) There was enlargement and thickening of the epidermis, especially of the hair follicles, which usually contained plugs of hornifying substance. Histologically the growths were similar to those described by Itchikawa and Baum, and which they term folliculo-epitheliomas. Tarring of the rabbits was discontinued after the sixth month.

As time went on and no results of the tar application on the goats could be noticed, it was thought that the area of tar absorption was too small. The ear of the goat or the perineum presents about the same surface as the ear of a rabbit. For this reason it was decided to increase the surface of absorption by painting additional areas on the body. This was done in the case of Nos. 15689, 15690, and 9983, but without any effect on the action of tar on skin. In these cases, and also No. 17301, the tarring was pushed to its toxic limit as evidenced by the rapid emaciation following on extensive tar application.

After a continuous period of tar application extending over 15 months, during which no alteration in the treated skin can be noted, one is forced to the conclusion that the Angora goat is highly refractory to the carcinogenic action of coal-tar.

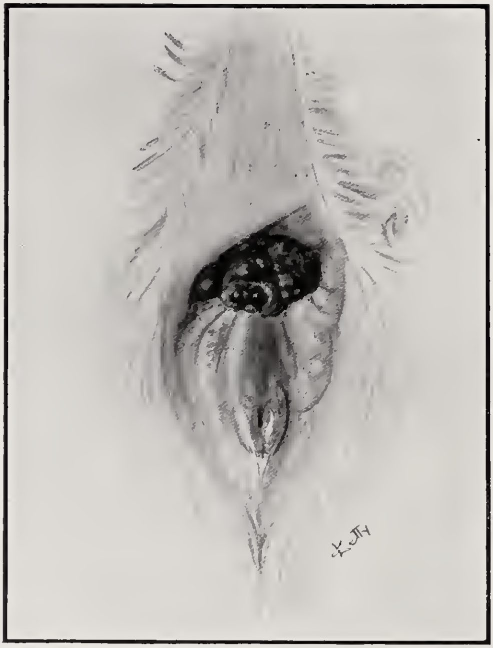
It would be most interesting to know to what factor this resistance might be ascribed, since this animal is naturally susceptible to spontaneous skin cancer.

Fig. 1.



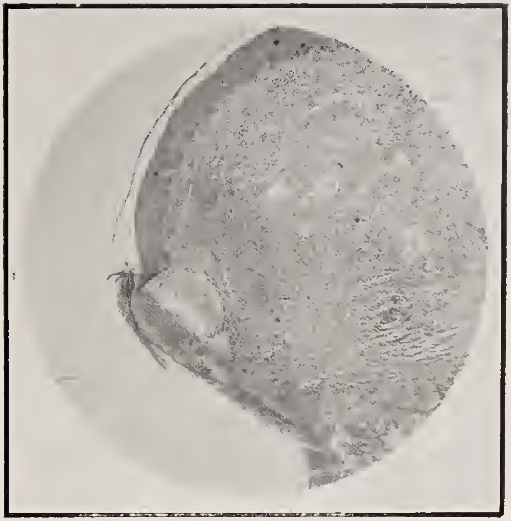
14771. Anal tumour. 1.9.26. Basal cell carcinoma.

Fig. 2.



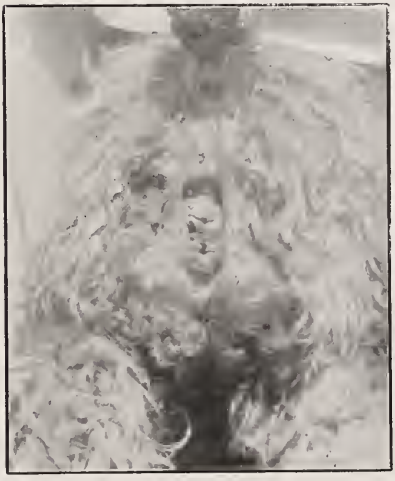
14771. From drawing two weeks later. Regression due to necrosis.

Fig. 3.



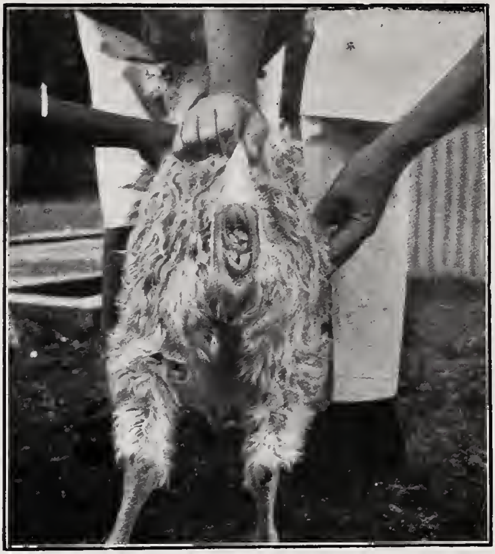
14771. Focus of little differentiated epidermal cells at junction of anal mucosa above and ulcerating bed of tumour below. 24×.

Fig. 4.



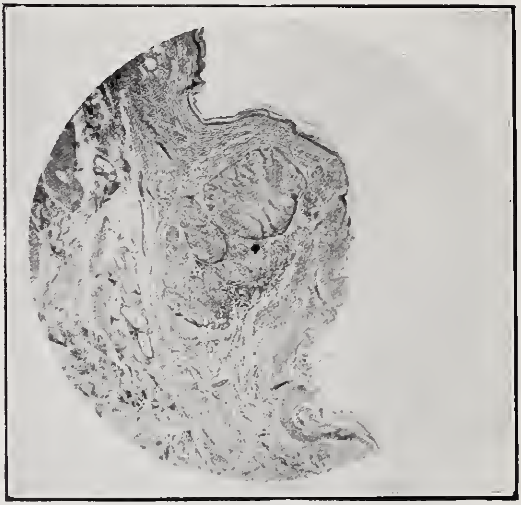
14772. Small tumour to left of anus on 1.9.26, before operation. Basal cell carcinoma.

FIG. 5.



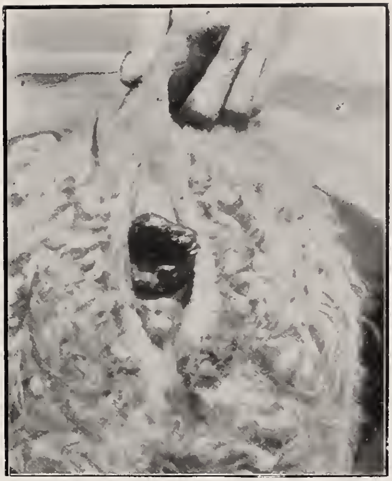
14772. The same fourteen months after operative removal of tumour. Note small excoriations under tail.

FIG. 6.



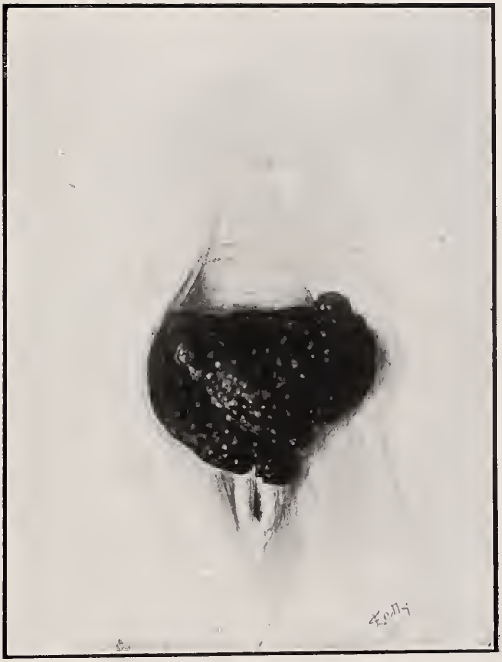
14772. Twenty-five months after operation. Focus of epithelial cells, presumably from the basal layer of sebaceous gland. Note atrophy of epidermis in parts. 24×.

FIG. 7.



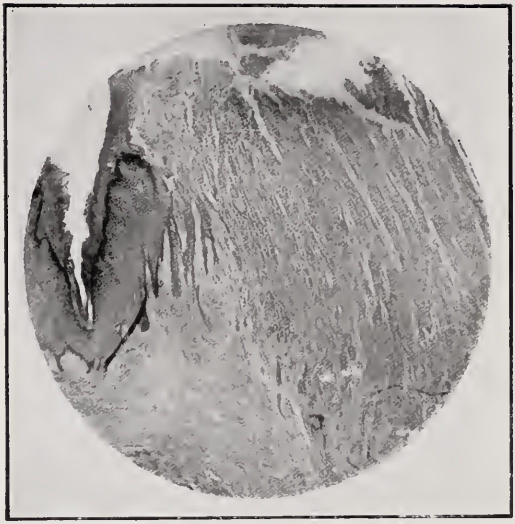
14773. Pigmented anal tumour. 1.9.26. Basal cell carcinoma.

FIG. 8.



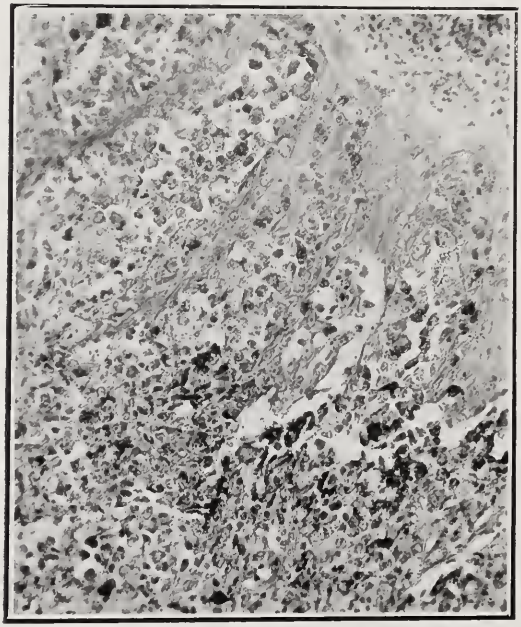
14773. The same from a drawing two weeks later, to show change in shape and size.

FIG. 9.



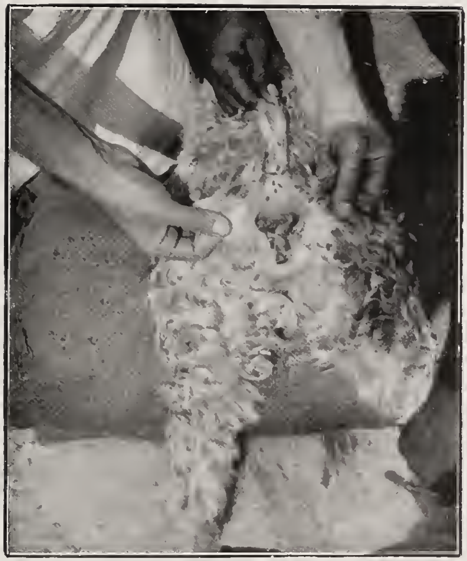
14773. Transition from the epidermis to tumour. 24×.

FIG. 10.



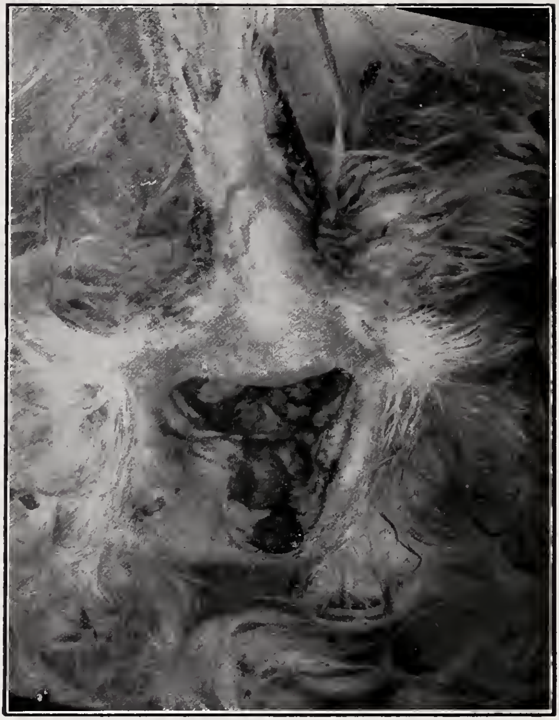
14773. Epidermis strands. The basal layer is indistinguishable from the tumour. Note the pigmentation. 200×.

FIG. 11.



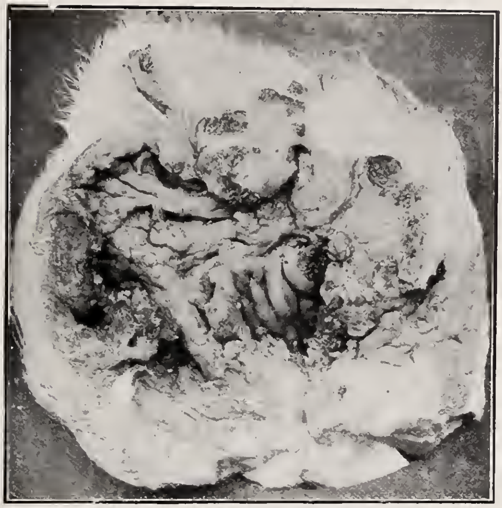
17293. Anal and vulva tumour.

Fig. 12.



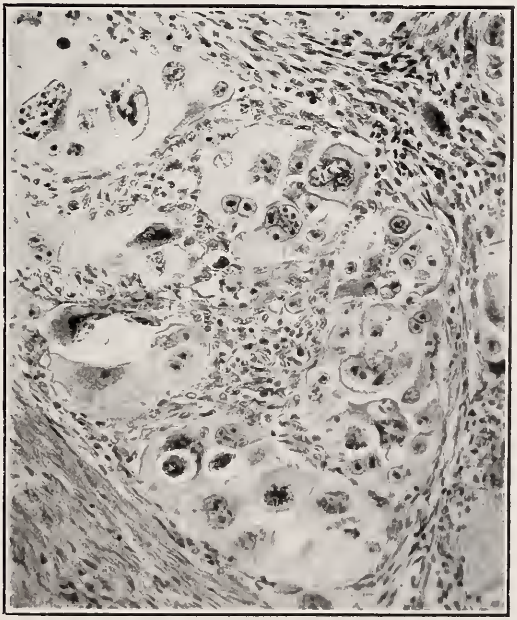
17293. The same. Closer view.

FIG. 13.



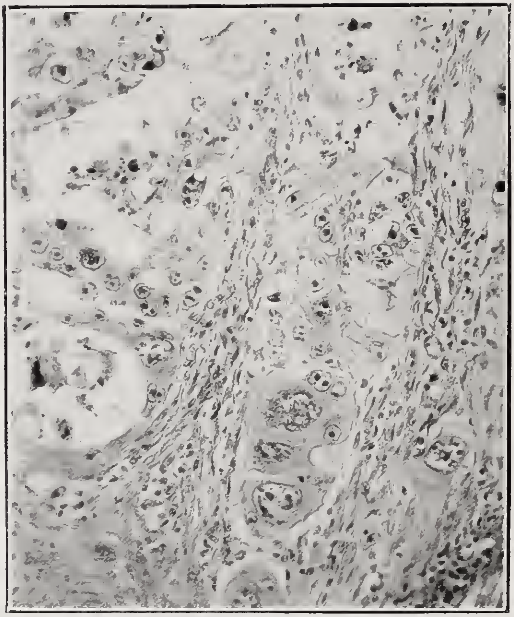
17293. Tumour after death of animal. The rectum opens on the floor of ulcer. Vulva is distorted. Spinous cell carcinoma.

FIG. 14.



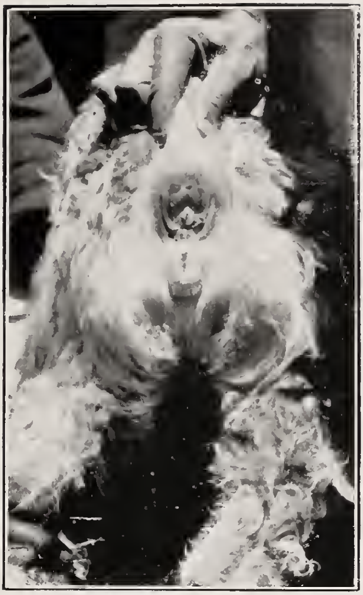
17293. Types of giant, degenerated, and keratinising cells found in above tumour. (" Cancroid parasites.") 200×.

Fig. 15.



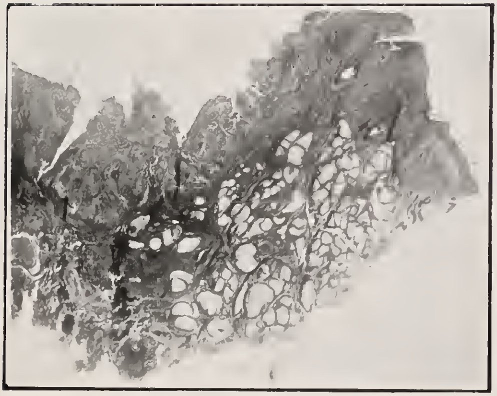
17293. The same as Fig. 14. 200×.

Fig. 16.



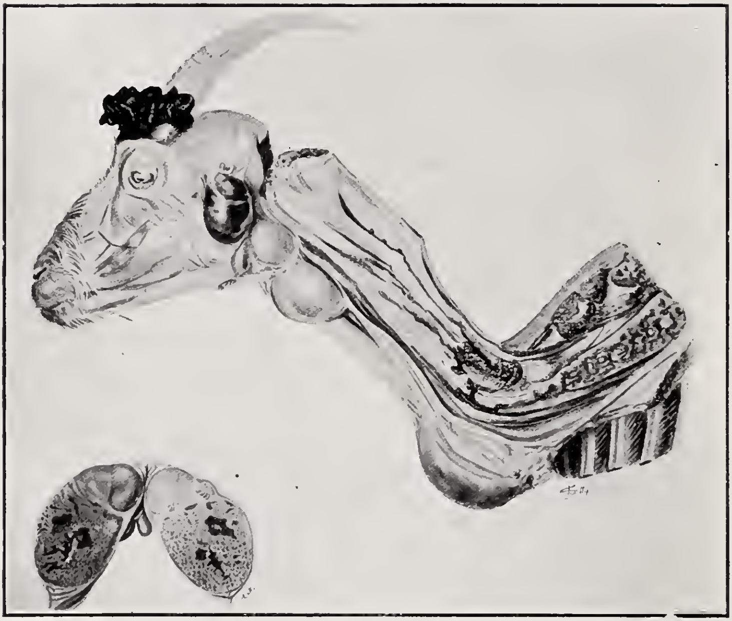
17294. Small tumours above anus. 2.11.27.

Fig. 17.



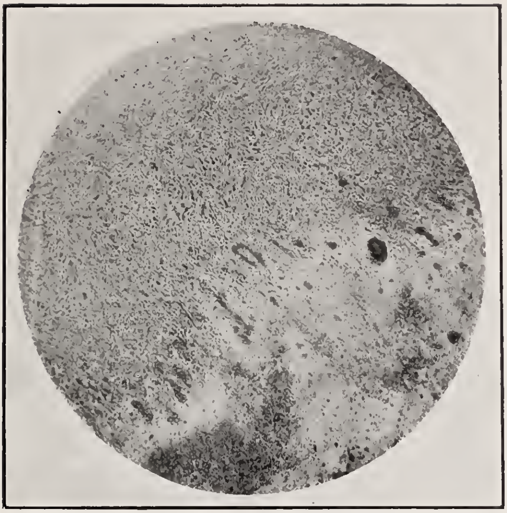
17294. Section through tumour removed surgically. Note the numerous sebaceous glands and the epidermis as it merges into the ulcerating part. 8×.

FIG. 18.



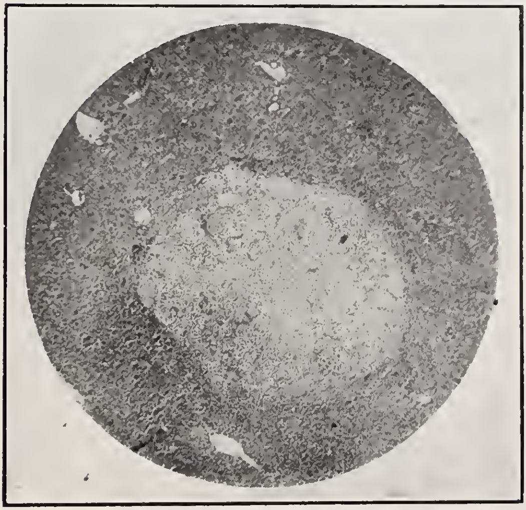
17296. Pigmented horn tumour with metastases in the lymphatic glands of head and neck. Inset, the prescapular gland in section (from drawing).

FIG. 19.



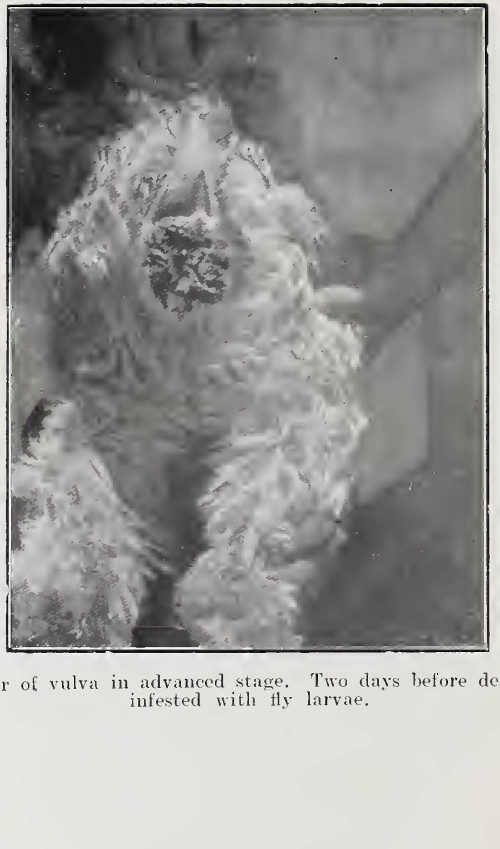
17296. Primary horn tumour. Vascular zone between necrotic surface and basal cell carcinoma proper. 24×.

FIG. 20.



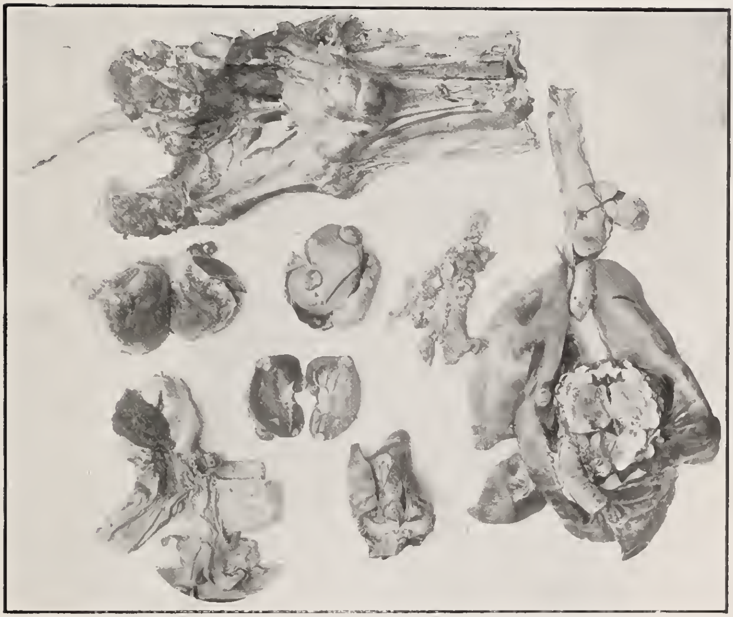
17296. The same. Metastases in the liver. 24×.

FIG. 21.



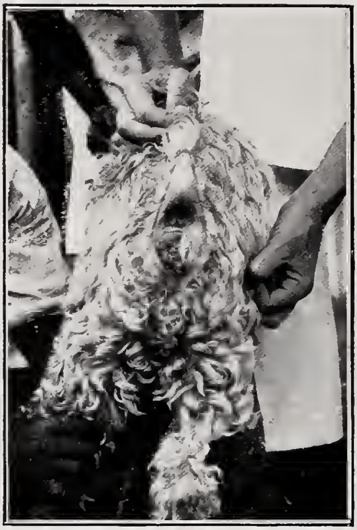
17297 Cancer of vulva in advanced stage. Two days before death. Heavily infested with fly larvae.

FIG. 22.



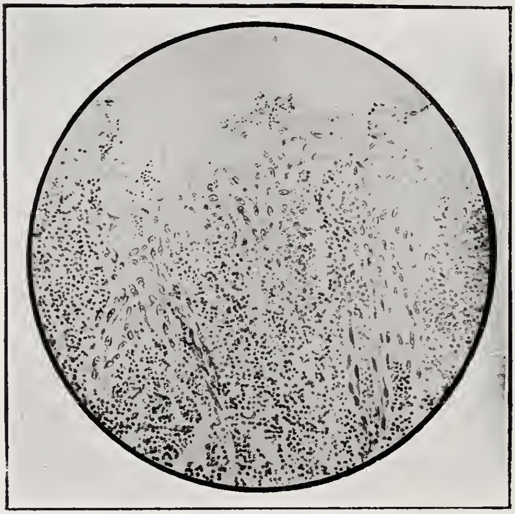
17297. Tumour after killing off fly larvae; note contraction. Secondary metastases in supramammary, sublumbar, bronchial, and mediastinal lymphatic glands, kidneys, heart, lungs, pancreas, and other organs.

FIG. 23.



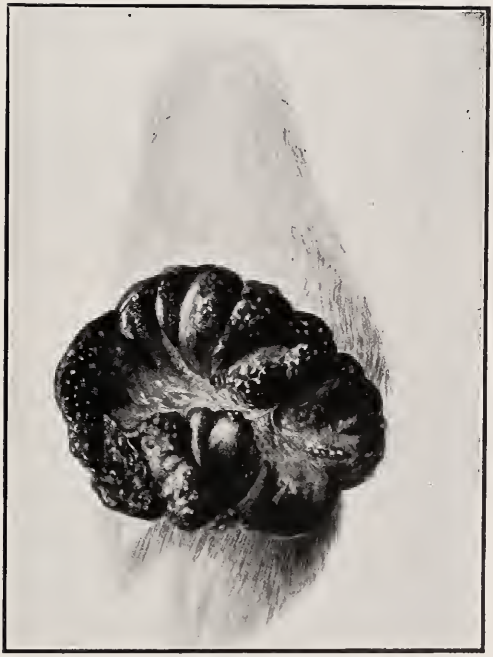
17298. Cancer of the vulva. Spinous-cell carcinoma. 10.11.27.

FIG. 24.



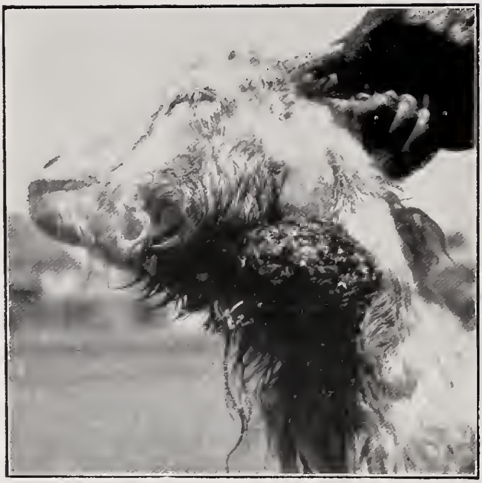
17298. Secondary metastasis in the supramammary lymphatic gland. Cancer cells penetrating the glandular tissue like roots. (From a drawing.) 170×.

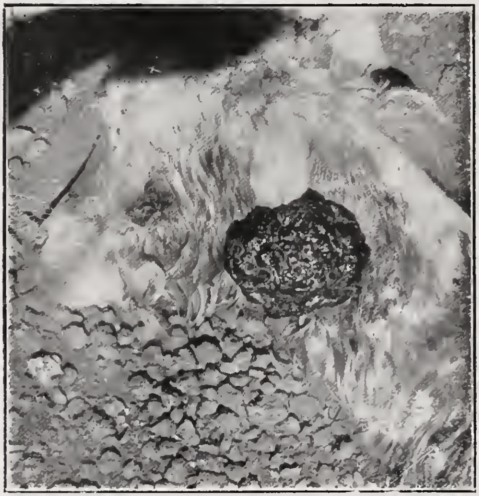
Fig. 25.



17299. Basal cell carcinoma of the ear. 5.7.27. (From a drawing.)

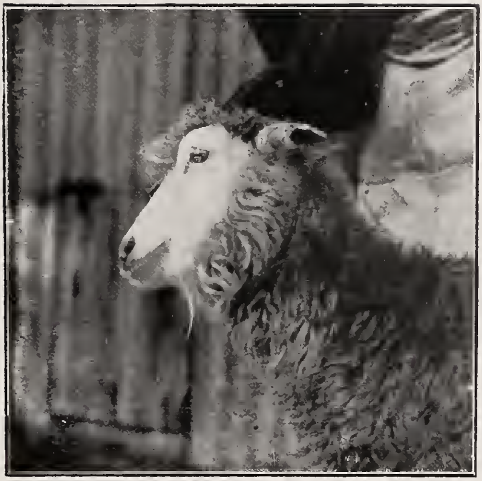
FIGS. 26 AND 27.





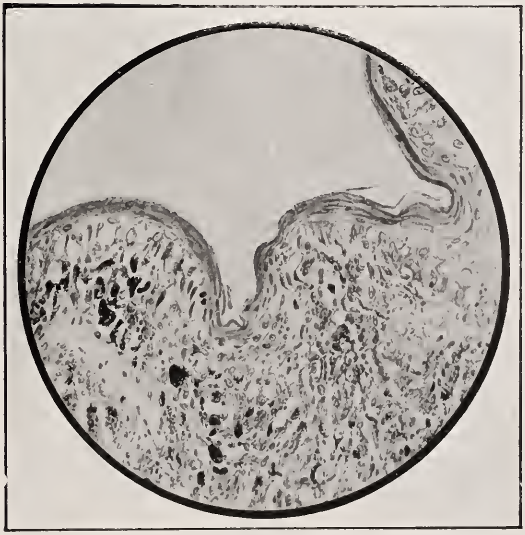
17299. The same on 28.9.27. Before operation.

Fig. 28.



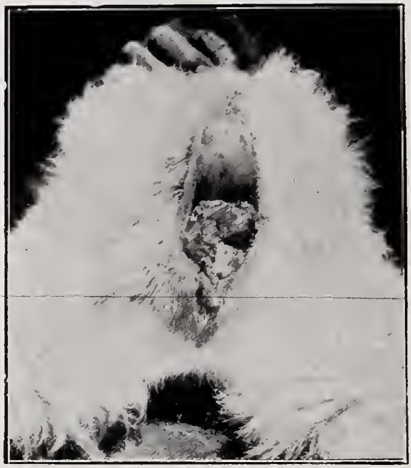
17299. The same goat after operation and recovery. 2.11.27.

Fig. 29.



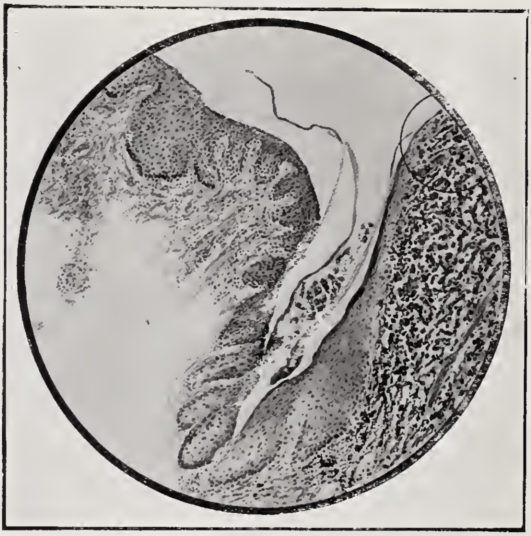
17299. Section of tumour. Note alteration in basal layer of epidermis and pigmentation. (From a drawing.) 170×.

Fig. 30.



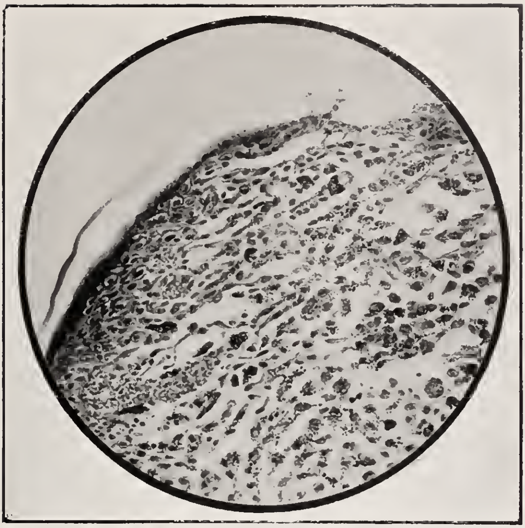
21957. Cancer of anus extending towards vulva. Mixed basal and spinous-cell carcinoma.

Fig. 31.



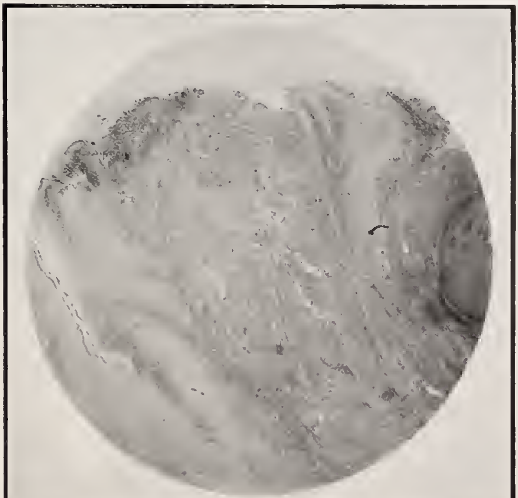
7256c. Epidermis going over to pigmented basal cell carcinoma. Note the patchy distribution of pigment in basal layer. 50×. (From drawing.)

FIG. 32.



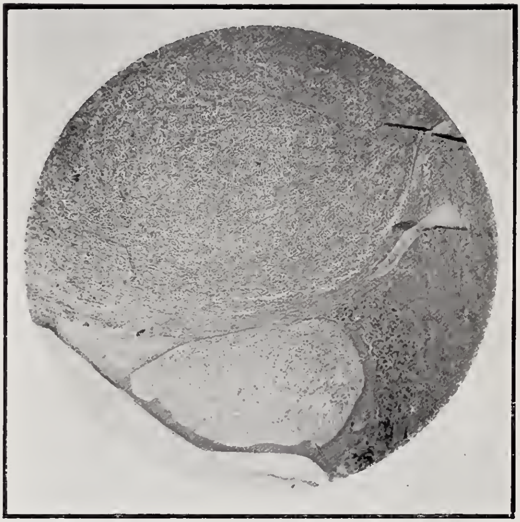
7256c. The same. Transition from epidermis to tumour tissue. Note pigmentation. 190×. (From drawing.)

FIG. 33.



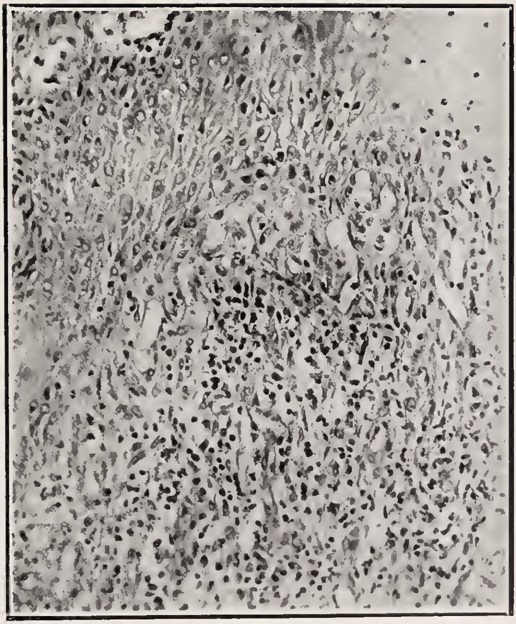
7256E. Crypt-like and tubule formation by rows of undifferentiated epidermal cells. 24×.

Fig. 34.



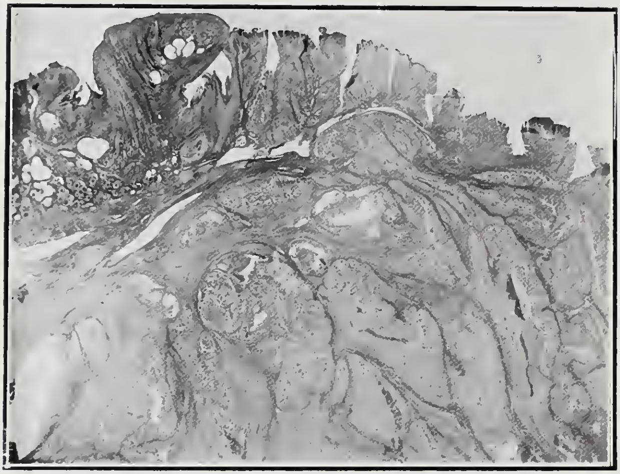
7256a. Typical structure of basal cell tumour in goats. Note uniformity, delicate stroma, and patchy pigmentation. 24×.

Fig. 35.



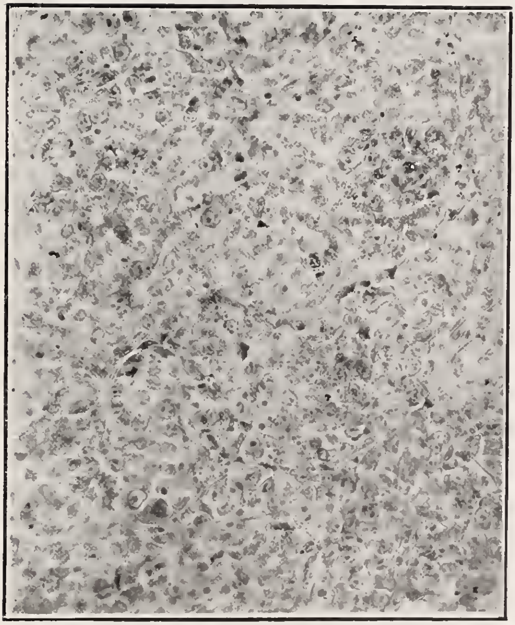
Basal cell carcinoma. 7256a. To show the transition between epidermis above and the loose tumour tissue in centre and below. 200×.

Fig. 36.



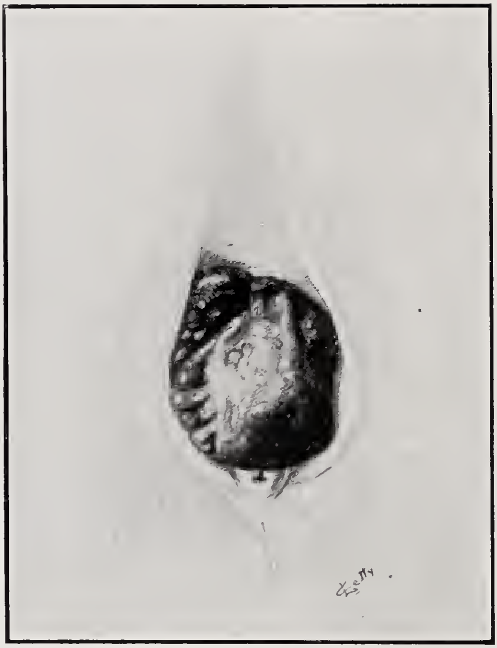
7256e. Basal cell carcinoma. To show the stroma and adenoid arrangement. 6×.

FIG. 37.



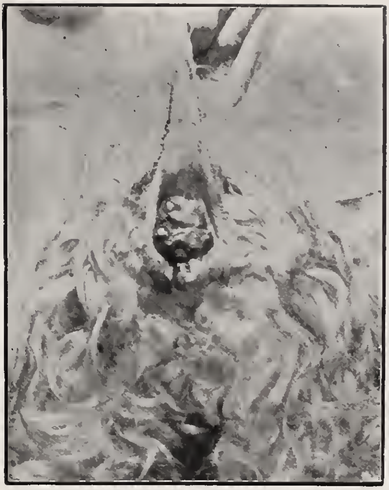
7256D. Basal cell carcinoma. Early stage. Closely packed epithelial cells with large round nuclei. Very delicate stroma. 200×.

FIG. 38.



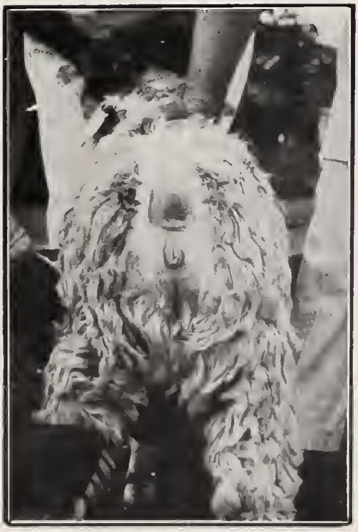
14770. Cancer of anus. Basal cell carcinoma? (From a drawing.)

Fig. 39.



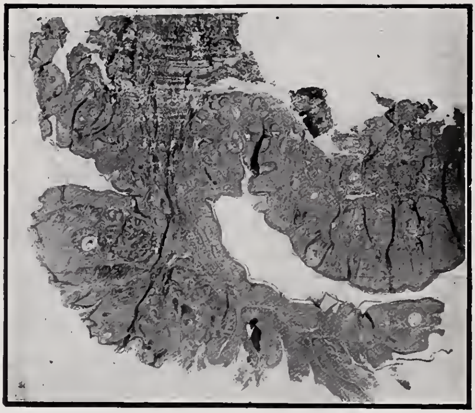
14770. The same. This tumour was eaten away by fly larvae.

Fig. 40.



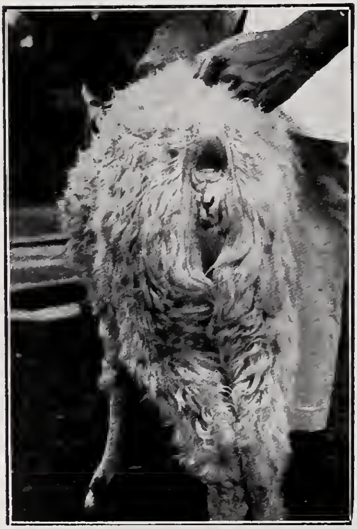
14770. The same animal after complete healing up of perineum.

FIG. 41.



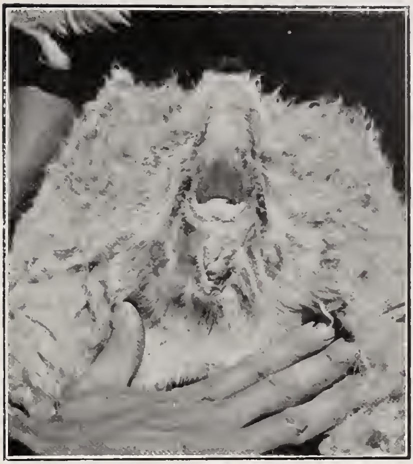
9983. Papillomatous growth. Note alternate atrophy and hypertrophy of epidermis. Also the proliferation of basal cells in the large sebaceous glands beneath epidermis. 8×.

FIG. 42.



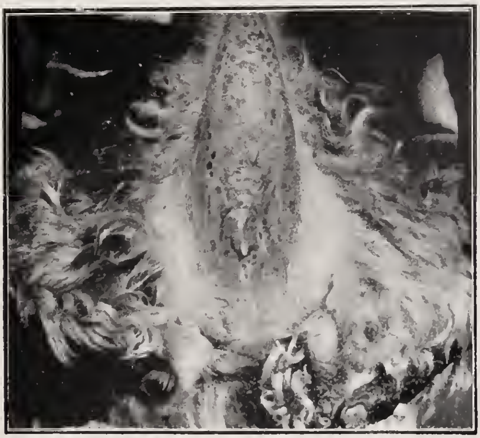
14505. Lacerated wound of vulva inflicted by horning seven months before.

FIG. 43.



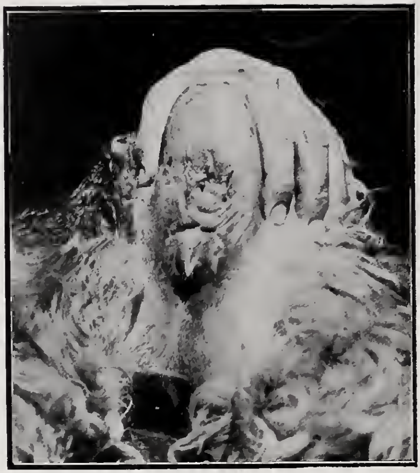
14505. The same, nineteen months after horning took place. No tendency to heal, slight swelling and superficial excoriations can be seen.

FIG. 44.



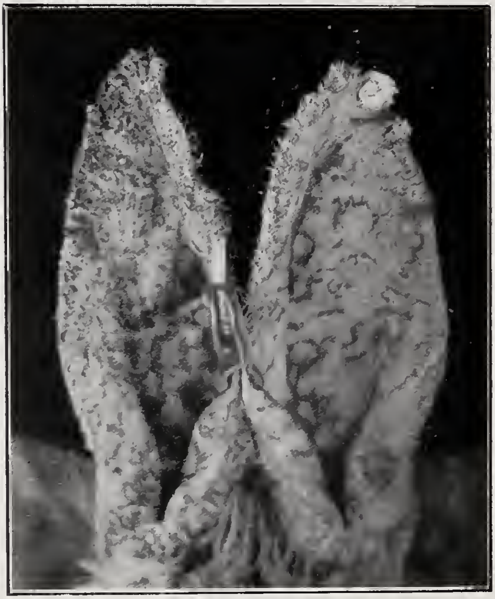
15690. To show pigmented patches of perineal skin.

FIG. 45.



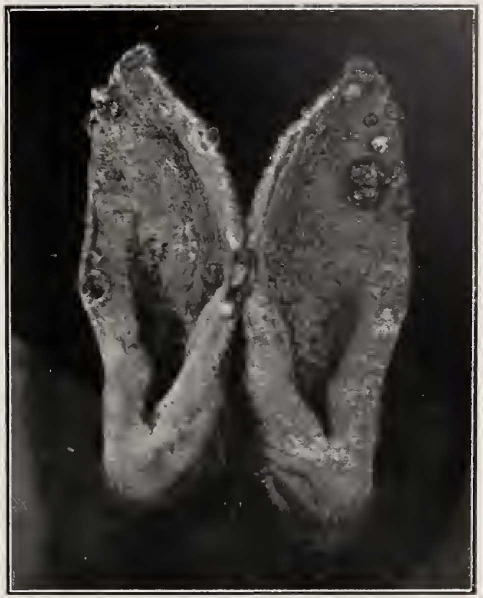
15690. Skin of perineum stretched out to show pitted appearance in glandular region and the small epidermal excrescences sometimes seen.

FIG. 46.



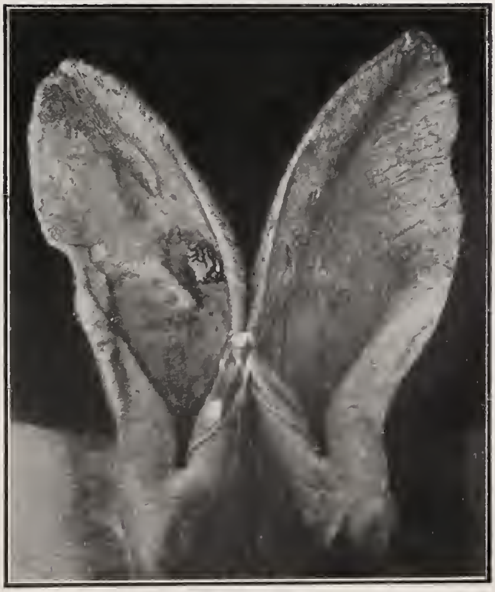
Rabbit No. I

FIG. 47.



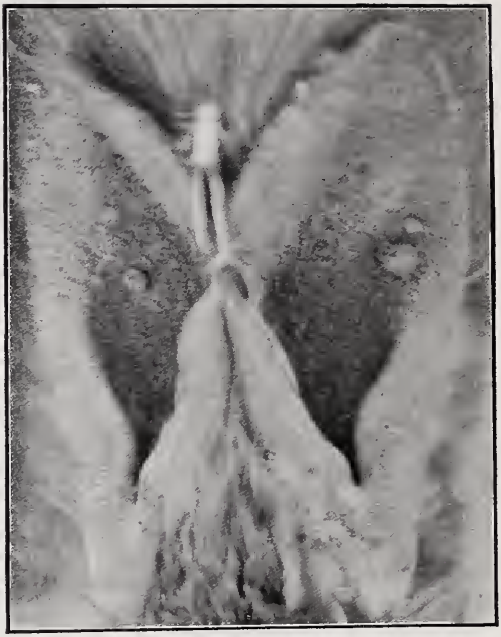
Rabbit No. II.

FIG. 48.



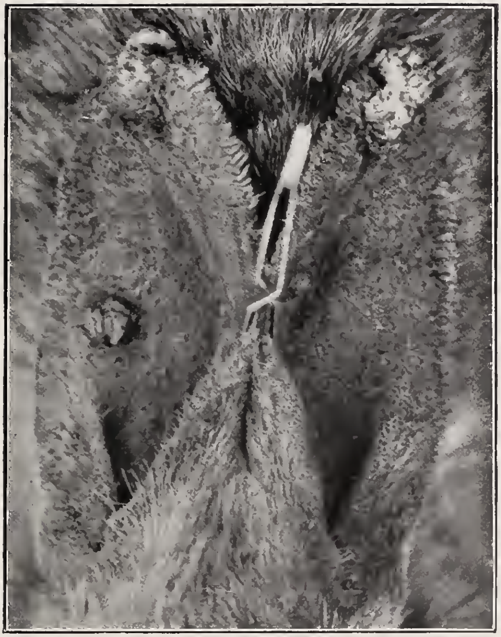
Rabbit No. III.

Fig. 49.



Rabbit No. IV.

FIG. 50.



Rabbit No. V.

Figs. 46-50.—Hornifying papillomatous growths (*folliculo-epithelioma*) resulting from painting with coal-tar. Goats painted with the same tar on the same dates showed no change in the skin, even after tar applications during twelve months and over.

Section VI.

Diseases due to Poisonous Plants.

J. I. Quin	Further Investigations into Geeldikkop (*Tribulosis ovis*).
J. I. Quin	The Toxic Properties of *Cucumis myriocarpus* Naud. and *Cucumis africanus*, Linn.
D. G. Steyn	Recent Investigations into the Toxicity of known and unknown Poisonous Plants in the Union of South Africa.
C. P. Neser	Vlei Poisoning, Part I.
A. O. D. Mogg ...	Vlei Poisoning, Part II.

15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

Further Investigations into Geeldikkop (*Tribulosis ovis*).

By J. I. QUIN, B.V.Sc., Veterinary Research Officer, Onderstepoort.

INTRODUCTION.

As mentioned before (*Jl. S. Afr. Vet. Med. Assoc.*, 1928), the problem of Geeldikkop in small stock in large areas of the Cape Province and Orange Free State is still a serious menace to stockowners. Although the actual prevalence of the disease is restricted to less than three months of the year, the losses during that time may be enormous, not only due to actual deaths, but to the suffering and loss of condition and wool in chronic cases.

Compared with the past season, the present one seems to be mild, since, although the disease is again widespread, the number of animals affected and the actual deaths recorded are much below those of the previous season.

Experiments were conducted in the Burghersdorp District on the southern bank of the Orange River, where a fairly severe outbreak had occurred on the farm Elswald. When experiments were started in the middle of January, *Tribulus terrestris* was found growing in abundance and with all the different stages of growth present. Merino lambs 7-12 months old were grazed in paddocks erected on—

(*a*) young preflowering Tribulus;
(*b*) flowering Tribulus;
(*c*) late-fruiting Tribulus.

The animals were only allowed the Tribulus and nothing else.

True geeldikkop cases made their appearance simultaneously on all three stages of growth, and in approximately equal proportions. The first cases appeared early on the third day, while by the sixth day, of the nine lambs placed in the different experiments, eight had developed dikkop. Soon after these cases had appeared heavy rains set in and by the 23rd January approximately four inches had fallen. When a larger number of lambs, despatched for this experiment from the western Free State, were now placed in the various experiments, not a single case of geeldikkop could be produced, although they were kept only on Tribulus for eight days.

The disease had undoubtedly made an abrupt disappearance between the 22nd and the 26th January.

DRENCHING EXPERIMENTS.

Drenching experiments were also resorted to, in which the juice expressed from the whole plant in the green late-fruiting stage was found to cause death of sheep within one to two hours. The juice from 2 lb. green plant and less produced no effects, while that from 3 lb. and more caused rapid death. No symptoms of geeldikkop, however, were noticed; as a matter of fact, animals seemed to die without showing any symptoms. On post-mortem no pathological changes were noticeable except that the whole volume of blood was changed to dark brown chocolate colour, with hardly a tinge of red in it.

On the day that the last case of experimental geeldikkop had appeared, a large amount of the late-fruiting Tribulus was pulled out and made into hay, being dried in the shade. When dried, the leaves and fruits were shed from the stems. Further experiments with the dry leaves and fruit were conducted at Onderstepoort.

Case A.—A Merino, 12 months old, was dosed twice daily with ½ lb. of the dry, finely powdered Tribulus hay for three days without any marked symptoms except slight tympanitis.

Case B.—A full-mouth Merino hamel was dosed with the watery extract prepared by soaking 650 grams of the powdered hay in three litres of water overnight. The animal died suddenly 75 minutes after dosing. No symptoms were noticed. On post-mortem the only change noticeable was again the marked discolouration of the blood, which appeared a dark, chocolate brown and tarry in nature. The visible mucous membranes were likewise of a brown colour.

Case C.—Merino lamb, 12 months old, was drenched with watery extract prepared from 420 grams dry Tribulus hay. The animal died 90 minutes afterwards. Fifteen minutes before death the first symptoms were noticeable. The animal had stopped feeding and was standing quietly with the head held in a corner of the stable. Respirations were slightly shallower and faster than normal. When forced to move the animal walked with a swaying and staggering gait and appeared especially weak in the hind-quarters. At the same time there was a progressive darkening and brown discolouration of the conjunctiva. About five minutes before death the animal fell down and passed into a state of coma, with twitching of the muscles of the hind legs accompanied by jerky flexing and extending of the limbs. The cutaneous reflexes were markedly accentuated. Respiration now became very deep and much slower than normal. With each expiratory movement there was a prolonged groan. The nostrils showed marked movement. The heart-beat, which at first was accelerated, now became faint and slow. Death took place within 15 minutes after the first symptoms had been noticed.

On post-mortem, no macroscopic changes were noticeable, except for the extensive dark chocolate brown discolouration of the blood, which also was reflected in the colour of the lungs. Spectroscopic examination of the blood in weak dilution revealed a fairly distinct absorption band in the red between the C and D lines. A drop of ammonium sulphide added to this blood dilution immediately changed it to a bright red colour, showing the typical bands for oxyhaemoglobin. All evidence thus seems to point to a toxic principle present in the watery extract of the Tribulus which causes rapid transformation, perhaps by oxidation, of haemoglobin into some chemical compound such as methaemoglobin, in which case the respiratory function of haemoglobin is rapidly decreased or lost. This naturally must lead to death from asphyxia or oxygen starvation. The process, however, seems to be reversible, since rapid recovery and disappearance of the brown colour is often noticed.

Aqueous extract of Tribulus hay when added in small amounts to either washed suspensions of sheep's corpuscles or weak dilutions of haemoglobin, causes the appearance of the brown colouration within one hour and with absorption bands the same as that produced by the poisoned sheep's blood.

Whether this toxic principle stands in any relation to the aetiology of geeldikkop is as yet unknown. It, however, seems possible that the formation in the cell body of a compound such as methaemoglobin may render some of the cells useless for further respiratory functions. Such cells may then undergo phagocytosis, and through transformation of the pigment give rise to the peculiar and intense yellow discolouration of tissues usually seen in geeldikkop. How actually the oedema is caused in geeldikkop is another point not settled at the moment. It still seems more than likely that some climatic factor, e.g. photosensitization, plays a rôle, seeing that the oedema is strictly localized to the bare and exposed parts of the head. Ultra-violet-ray treatment in conjunction with Tribulus feeding is being carried out at the moment and may possibly throw further light on this elusive problem.

Dr. de Kock, who has examined pathological sections from geeldikkop cases, maintains that the pigment found in sections is not typical of the pigments associated with abnormal blood destruction, nor is its distribution similar. The pathology is being fully studied and a full report will be submitted in due course.

I here wish to tender my thanks to Dr. de Kock for help and advice given and also to Mr. I. D. Steyn, "Elswald," district Burghersdorp, for kind help given, and facilities granted while conducting experiments on his farm.

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15th Annual Report of the Director of Veterinary Services, Union of South Africa. October, 1929.

The Toxic Properties of *Cucumis myriocarpus* Naud., and *Cucumis africanus* Linn.

By J. I. QUIN, B.V.Sc., Veterinary Research Officer, Onderstepoort.

As is the case with several other wild growing species of *Cucumis*, the two species *Cucumis myriocarpus* and *Cucumis africanus* are found widely distributed throughout South Africa. Both these species are green, annual herbaceous plants with succulent prostrate stems. The fruits (gourds) of *C. myriocarpus* when ripe are about the size of small walnuts, brownish or yellowish, and covered with soft fleshy prickles. In the case of *C. africanus*, the gourds are much larger, being about the size of a hen's egg and of a pale lemon-yellow colour. In both cases the internal structure of the fruit is the same as that of the ordinary cucumber, the juice emitting a strong cucumber odour. The green fruit is of a slightly bitter taste, which, however, especially with *C. myriocarpus*, becomes markedly accentuated as maturity is reached, while the seeds are of an oily, not unpleasant, taste. Both species may be found growing wild in the open veld, although *C. myriocarpus* prefers cultivated soil; for example, it is frequently found growing as a weed in mealie lands. (For detailed description of these species, see *Flora Capensis*, Vol. 2, pp. 494-496.)

Various samples of the fruit of *C. myriocarpus* have been submitted for identification and report as to their toxicity, as stockowners suspect it to be the cause of death among small stock, especially sheep grazed on mealie lands during the winter months. It is reported that during that time, when other succulent green food is scarce, sheep may be seen eating these " bitter apples " or " wild cucumbers," as they are popularly termed, along with such other juicy fruits as that of the wild gooseberry (*Physalis minima*). Death frequently takes place without marked symptoms and with the aetiology left unexplained. Various experiments have accordingly been conducted with the fruit of *C. myriocarpus* sent in. On no occasion were the fruits of *C. africanus* submitted and thus presumably not suspected to be toxic. Fruits of this species were therefore collected locally for comparative tests with *C. myriocarpus*.

FEEDING TESTS.

As the well-nourished experimental animals are disinclined to ingest fairly large amounts of the material within short intervals, drenching of pulped fruit by means of the stomach-tube was resorted to. The material drenched consisted either of the fruit juice in the natural state or the dry pulverized juice dissolved in water. It was soon noticed that in both species the green fruits were far less toxic than the matuie ones, being also less bitter.

In the case of *C. myriocarpus*, the following amounts proved to be the approximate minimal lethal dose for the various animals:—

(*a*) *Guinea-pigs.*—Juice from 10-12 grams fruit killed within six to nine hours.
(*b*) *Rabbits.*—Juice from 15-20 grams fruit killed within about nine hours.
(*c*) *Sheep.*—Juice from 1 kilogram ripe fruit killed in about six hours, while the juice from 2½ kilos green fruit only produced a mild diarrhoea.
(*d*) *Dogs.*—The juice when given by the mouth is rapidly vomited, although severe symptoms of haemorrhagic diarrhoea may persist for several days.
(*e*) *Horse and Bovine.*—Four kilograms drenched to each did not prove fatal.

The seeds and rinds when washed free from adhering juicy material proved to be completely atoxic, as 50 grams of each dosed to rabbits had no effect.

With reference to *C. africanus*, the same effects were produced on guinea-pigs and rabbits, except that the juice of approximately twice the amount of ripe fruit was needed to kill, whereas sheep, cattle, and horse could not be killed even though several kilograms were given.

EXTRACTION OF TOXIC PRINCIPLE(S).

(*a*) *From the Natural Juice.*—A highly toxic substance can be extracted from these species by the following process:—

A large amount of organic matter is precipitated from the juice by alternate addition of basic lead acetate and filtration until no further precipitation occurs. The excess of lead in the filtrate is removed by adding an aqueous saturated solution of sodium carbonate and again filtering until all the lead is removed.

The clear filtrate is now shaken up with an equal volume of ether in a separating funnel. This is repeated two or three times and the etherial portion evaporated to dryness. The residue is then found to consist of a white powder, which at first appears crystalline, although, on close examination, it is seen to be amorphous and with a glassy lustre. This white powder is intensely bitter and of a peculiar bitter odour. In this state it is only sparingly soluble in water, although readily so in alcohol, ether, chloroform, and acetone. On heating it changes to a resinous sticky mass. Attempts at further purification and crystallization have so far been unsuccessful. It may, however, be definitely stated that this powder is non-alkaloidal and non-glucosidal in nature, and further that it is nitrogen-free. From its general characteristics it appears to be either a true resin (one or more) or closely associated with a resin, or, thirdly, that it may undergo resinification in the process of extraction. On an average the ripe fruit of *C. myriocarpus* is found to yield .04 per cent. of this powder, while in the unripe state the yield is much less. In the case of *C. africanus* the final product obtained appears to be identical in all respects except that the yield is only one-third to one-half that obtained from *C. myriocarpus*. Further, when tested out weight for weight, the powder from *C. africanus* is as toxic as that from *C. myriocarpus*, while the symptoms and lesions produced are identical. The aqueous residue left after extraction with ether is faintly toxic on account of traces of the poison left behind in the dissolved ether.

(*b*) *From the Dry Powdered Juice.*—The same powder can be obtained from the dry powdered juice when treated as described above for the fresh juice. Comparative tests carried out showed that the toxicity was fully maintained after desiccation of the juice.

EFFECTS OF DOSING.

As mentioned before, death takes place suddenly without characteristic symptoms preceding it. According to stockowners, sheep which in the evening appear to be normally healthy are found dead the next morning. On opening the carcase, large numbers of the undigesed seeds are found in the gastro-intestinal tract.

When dosed, both the juice and the powdered extract may cause sudden death, especially when given in fairly large amounts. With smaller doses a diarrhoea of varying severity is noticed.

Post-mortem Lesions.—This depends on the dose given. With smaller doses, the main lesions are found in the gastro-intestinal tract. All the blood-vessels in the splanchnic area are markedly injected and show up prominently. There is a severe hyperaemia of the gastro-intestinal mucosa which may in some cases give rise to the formation of a croupous pseudomembrane over large areas, accompanied by haemorrhage into the lumen of the intestine. As a rule, however, varying amounts of what appears to be whole coagulated blood plasma may be found in the pyloric portion of the stomach and initial portion of the small intestines. In such cases the blood is markedly concentrated and cyanotic in appearance. Where large doses are given, the animal usually dies as the result of an acute pulmonary oedema, the gastro-intestinal lesions being less marked.

RESULTS OF INJECTION OF CUCUMIS EXTRACT.

The result of the injection of the powdered extract is perhaps the most interesting and the most significant phenomenon to be recorded. Due to its low solubility in water, the powder is usually injected in weak (5-10 per cent.) alcoholic solution. With the exception of the horse which could not be killed by injection, all the species of animals used, proved highly susceptible to the poison. With amounts higher than the minimal lethal dose death regularly took place in from one to two hours. Following intravenous injection, animals usually died slightly sooner than from the subcutaneous injection although the required dose is the same. Thus doses from .005 to .01 gram are regularly fatal to guinea-pigs and rabbits, while dogs and sheep are killed with amounts from .05 to .1 gram and bovines with .75 gram. With daily intravenous injection of sublethal doses no visible effect is produced even after a week. Subcuaneous injections, however, even with minute quantities, cause widespread and very severe oedematous swellings of the neighbouring parts. The swelling is not accompanied by fever, nor is it unduly hot to the touch. Furthermore, the overlying skin is not markedly changed in colour, i.e. there is no marked redness or signs of inflammation. On the contrary, the skin appears pale and lifeless. On incision into the swelling, the skin and subcutaneous and even intermuscular tissues are seen to be markedly thickened and infiltrated with a clear homogeneous viscid fluid, which rapidly coagulates on exposure. The swelling subsides gradually and healing is very slow, the skin and subcutaneous tissues being left in a thickened fibrous state. With lethal doses, on the other hand, whether injected subcutaneously or intravenously, a fulminating effect is produced. No characteristic symptoms are noticeable except a progressive cyanosis of the visible mucous membranes shortly before death, when signs of respiratory distress also become evident. Gurgling sounds may be heard over the thoracic region. At the moment of death copious amounts of clear white froth are forcibly extruded through the nostrils and even through the mouth. This again coagulates rapidly. In the case of dogs this expulsion of froth is frequently absent, while in the other species it is a practically constant feature. Dogs may, however, show marked diarrhoea and vomiting soon after injection.

Post-mortem Lesions.—The blood again is markedly concentrated, cyanotic, and slow in coagulating.

The lungs are fully dilated, with the pleura glistening and moist with a varying degree of hydrothorax present. The lungs are of a dark purple-red appearance. On section a copious amount of white froth and clear liquid resembling blood plasma issues forth from all bronchi and bronchioles, the parenchyma appearing saturated with liquid.

Apparently the liquid is held under high pressure, gauging from the rate that it flows off from a cut surface. The blood in the pulmonary vessels is thick, tarry, and markedly cyanotic. The vessels themselves (even the larger ones) are surrounded by a broad zone of clear watery material which coagulates rapidly on exposure.

The heart usually stops in diastole and is filled with dark-blue venous blood. Subendocardial haemorrhage in the left ventricle is of frequent occurrence. Hydropericard may be marked, especially in rabbits and dogs. Congestion of the liver and kidneys is constantly noticed. The gastro-intestinal tract may be normal except for injection of the mesenteric vessels. In dogs there may be marked hyperaemia of the mucosa of the small intestines. In cattle and sheep the post-mortem findings closely simulate those of severe cases of heartwater. In cattle, especially, the interlobular septa of the lungs are markedly infiltrated and thickened.

In rabbits it has been determined that at least half of the total blood plasma may in this way escape from the circulation into the lung parenchyma and air-passages.

The following data will serve to illustrate the suddenness and the severity of the oedema.

Case A.—Heifer, one year old.

Determinations made on samples of normal blood:—

Viscosity, 3.2.
Percentage volume red corpuscles, 20 per cent.
Red count, 3.56 millions.

Powdered extract prepared from 675 grams green fruit, i.e. well below the M.L.D. for cattle, was injected subcutaneously behind the shoulder.

Twenty-four hours afterwards a tremendous swelling had developed of the foreleg and surrounding parts. The animal was very lame on that leg.

Blood determinations then gave the following results:—

Viscosity, 3.95.
Percentage volume red corpuscle, 32.5 per cent.
Red count, 7.92 millions.

This showed that there was a definite concentration of the blood, through loss of liquid.

A few minutes after this determination had been made .4 gram of the white powdered extract was injected intrajugularly. The animal died within 50 minutes after injection. The blood now gave the following values:—

Viscosity, 6.6.
Percentage volume red corpuscles, 55 per cent.
Red count, 12.2 millions.

In this state the blood could hardly flow through a fair-sized hypodermic needle. Large amounts of clear fluid and froth appeared at the nostrils, and a severe pulmonary oedema was noticeable.

Case B.—Merino sheep, fullgrown.

The following normal blood values were obtained:—

Viscosity, 3.4.
Percentage Volume red corpuscles, 29 per cent.

The animal was then injected intrajugularly .1 gram Cucumis powder. The animal suddenly died 60 minutes afterwards with froth flowing from the nostrils. Blood collected at the moment of death gave the following figures:—

Viscosity, 9.4.
Percentage volume red corpuscles, 70 per cent., i.e. more than twice the concentration of the normal blood.

Case C.—Merino sheep, adult.

Normal blood values were:—

Viscosity, 2.9.
Percentage volume red corpuscles, 28 per cent.

.05 gram powdered extract was injected intrajugularly and blood determinations made every hour afterwards. There was no marked deviation from the normal. After 24 hours another injection of .05 gram was given. The sheep died within five hours with the blood values as follows:—

Viscosity, 8.
Percentage volume red corpuscles, 63 per cent.

There was a fair amount of froth from the nostrils. On post-mortem there was a definite pulmonary oedema, although the most marked finding was a severe hydrothorax of 1,200 c.c.

In several cases where lethal amounts were injected in one dose the blood values were studied at short intervals. In all cases it was found that the normal values were maintained until within a few minutes before death, when a sharp and sudden rise would be seen. In none of the numerous cases produced were there any signs of haemolysis, nor was a single red cell found in the oedema fluid. Microscopic examination of sections from the lungs revealed large numbers of red cells in the smaller pulmonary vessels and marked oedema in the alveoli and perivascular tissues. No histological changes were noticeable in the blood-vessels themselves.

Physiological Action on Isolated Organs and Tissues.

On the isolated heart of rabbits perfused with oxygenated Ringer-Locke solution to which small amounts of the powder had been added, no marked cardiac disturbance was produced. The rate as well as the excursions may show a decline, although the heart kept on beating for hours. Similarly no effects were produced on the uterus of a rabbit or a piece of small intestine suspended in normal saline, even when fairly large quantities of the powder were added. The irritability of frog's muscle and nerve was in no way found to be changed from the normal.

Summary and Discussion.

The two species *C. myriocarpus* and *C. africanus* were tested out on animals and found to be definitely toxic. This was especially found to be the case when ripe fruits were used. The toxic principle(s) are stable, as the poisonous juice could be desiccated and even boiled for long periods without destroying the toxic effect. When dosed to animals, the juice when given in sufficient amount is rapidly fatal, although the symptoms and frequently the post-mortem findings are not very striking.

A highly toxic substance could be extracted from both species as described before. Except for the yield, which in *C. africanus* is much less than in *C. myriocarpus*, the two substances appear identical in all respects. The poison is definitely non-alkaloidal and non-glucosidal and probably of a resinous nature. On injection it is rapidly fatal, death coinciding with a very severe pulmonary oedema. From the nature of its action, it is indeed very difficult to arrive at a definite and clear understanding of the different factors involved. Clinically as well as pathologically, no clue can be found to explain the various points. It would, however, appear that the poison is very readily taken up by the blood-stream even following subcutaneous injection, and, with the venous blood, passed through the heart into the pulmonary circulation. In the lungs with a multitude of minute blood-vessels, the walls of which consist of a single layer of delicate endothelial cells, gross injury is effected, the poison apparently acting in a direct way on the endothelial lining.

This assumption, if correct, would imply some physico-chemical disturbance in the cell body rather than some morphological change. Thus in no case was pulmonary haemorrhage noticeable, i.e. actual tearing of blood-vessels can be excluded, and yet the insult offered to the minute vessel wall must be very significant when large amounts of apparently whole plasma and containing such large protein molecules as those of fibrinogen, are capable of being forced through the vessel walls. There seems to be little doubt that this is a case of a primary pulmonary oedema, since the heart-beat remains normal until shortly before death, when asphyxial conditions set in. The possibility of vasomotor disturbances accounting for the oedema seems remote. Blood-pressure tracings obtained from decerebrate dogs and rabbits show practically no deviation after intravenous injection of extracts. In such carcases the respiration and heart-beat come to an end from the same cause, i.e. the marked pulmonary oedema. When this appears there is a marked fall in the general blood-pressure, but not until this moment.

With small animals as much as half the plasma may be lost in this way, while in sheep a concentration of the red corpuscles from a normal of ± 25 per cent. to 75-80 per cent. is frequently noticed.

Death thus appears to result from a rapid filling of all the bronchi, bronchioles, and alveoli with plasma, so interfering with respiration. At the same time the marked concentration of the blood and the imperfect oxygenation must necessarily cause grave injury to vital tissues and organs such as the brain and heart.

The poison, therefore, appears to exert a marked injurious influence on the endothelial lining of minute vessel walls, so causing a sudden and marked increase in permeability.

I wish to record my thanks and appreciation to my colleague, Dr. G. de Kock, for examining the pathological sections; to Mr. A. O. D. Mogg, Ecologist in the Division of Botany, for identifying and submitting species of *Cucumis;* to Mr. M. Carlisle for capable assistance rendered especially with the physiological experiments; and to several farmers who have supplied information and submitted material.

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15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

Recent Investigations into the Toxicity of known and unknown Poisonous Plants in the Union of South Africa.

By D. G. STEYN, B.Sc., Dr.Med.Vet., Veterinary Research Officer, Onderstepoort.

It is proposed to record the above information in the alphabetical order of the names of the plant families. The procedure adopted in these investigations was, at first, to attempt feeding-experiments in which the plant under investigation was presented to the experimental animals either as such, or minced and mixed with forage, bran, bread, or mealie meal. In most cases this method was a failure, as practically all the animals, being accustomed to the laboratory foodstuffs, refused to take the plants unknown to them; consequently a lot of time was wasted. Drenching by means of a stomach-tube is now employed, as this method of experimenting with poisonous plants furnishes the most reliable and accurate information, although for different reasons it is not always practicable.

The following conditions may give rise to plant poisoning in animals grazing under natural conditions:—

(*a*) Under drought conditions, the animals, in the absence of edible vegetation, are forced to feed on poisonous plants. Typical examples are the "vermeerbossies" (*Geigeria* species) and "duwweltjie" (*Tribulus terrestris*).

(*b*) It very often happens that there is an intense intergrowth between the normal veld vegetation and poisonous plants, so that the animals are unable to avoid the ingestion of the latter plants and accidental poisoning occurs. Striking examples of this intergrowth may be seen in the "vermeerbossie" areas.

(*c*) Plant poisoning very often occurs in transport animals, as these hungry animals, when outspanned, partake greedily of practically any plant.

(*d*) Imported animals, or animals moved from one environment to the other, meet with poisonous plants unknown to them. Acclimatization, therefore, not only implies the immunization of animals against infectious diseases, but also a development of a sense of discriminating between edible and poisonous plants.

(*e*) Accidental poisoning may take place when hay contains poisonous plants. In the past this has very often been the case with Chinkerinchee and Crotalaria poisoning in horses.

(*f*) It is a well-known fact that aphosphorosis causes a craving for material which animals, under normal circumstances, will not ingest. This condition may also prompt animals to partake of poisonous plants.

(*g*) In the United States of America, the opinion is held that certain animals acquire a craving for poisonous plants, and this is then copied by others. These conditions may furnish one explanation for the sudden outbreaks of plant poisoning on farms on which such outbreaks have been unknown.

APOCYNACEAE.

NERIUM OLEANDER, Linn.

Synonyms.—Ceylon rose, oleander.

This plant is a native of the Levant, and is cultivated as an ornamental tree in South Africa. In America the leaves were proved toxic.

Origin.—J. J. Edgar, Goodwill, Uitenhage.

It proved to be toxic to rabbits, the M.L.D. of the dried leaves and flowers per kilogram body weight being five grams.

Symptoms.—Within ten minutes after dosage the rabbits showed restlessness, laboured breathing, and an extremely accelerated pulse, which later on became imperceptible. Death occurred within three-quarters of an hour after dosage. The plant has a digitalis-like action and death is caused through heart failure.

Post-mortem Appearances.—Marked cyanosis, hydroperitoneum, pronounced hyperaemia of the lungs and liver.

ARALIOCEAE.

CUSSONIA SPICATA, Thunb.

Synonyms.—Kiepersol, sambreelboom, cabbage-tree, nooisboom.

Origin.—Government Veterinary Officer, Grahamstown.

A sheep was drenched with 200 grams of the above leaves without any deleterious effects.

AMARYLLIDACEAE.

VALLOTA PURPUREA Herb.

Synonyms.—Berglelie, George lily, Knysna lily.

Origin.—Hall and Sons, Ltd., P.O. Tomango, Transvaal.

History.—There are no previous records as regards the toxicity of this plant.

The above owners lost a number of calves which had partaken of the bulbs of this plant, which had been dug out of a land and thrown over the fence.

Toxic Principle(s).—The toxin is alcohol and water soluble, and is only partially destroyed by ten minutes' boiling. The toxicity had not decreased to the slightest extent after ten months' storage. Various attempts to isolate the toxin met with no success.

Symptoms.—Rabbits, dogs, and sheep were drenched with the bulbs, and guinea-pigs were injected with extracts prepared from the bulbs.

Dogs drenched with 100 grams of bulb, vomited within five minutes, remained apathetic for a day or two, but ultimately recovered. The symptoms in sheep and rabbits were those of an irritant poison. The M.L.D. for sheep is approximately 150 grams, and for rabbits about 20 grams.

The symptoms appear four to six hours after dosage. There is a pronounced diarrhoea, which sets in about eight hours after dosage, and a consequent general weakness. The respiration is laboured and the pulse exceedingly accelerated. The affected animals die in a state of complete exhaustion from fifteen hours to a few days after dosage.

Post-mortem Appearances.—Marked general cyanosis, stasis in the subcutaneous blood-vessels, the blood is partly coagulated, very dark red in colour and tarry consistence, haemorrhagic swelling of the bronchial and mediastinal lymph glands, numerous subepicardial and subendocardial haemorrhages, marked hyperaemia and slight oedema of the lungs, marked hyperaemia and degeneration of the liver, marked acute catarrhal gastro-enteritis with haemorrhages.

Pathological Anatomical Diagnosis.

Liver: Extensive fatty changes, more marked round periphery of lobulus.

Kidney: Pronounced hyperaemia and fatty degeneration of some tubules.

Myocard: Extensive uniform fatty degeneration.

Lymph Glands: Acute haemorrhagic lymphadenitis.

NERINE SPECIES PROBABLY *N. MARGINATA*.

Origin.—P. L. le Roux, Pokkraal, P.O. Rawsonville, Worcester.

History.—There are no previous records of its toxicity.

Symptoms.—The seeds, which bear a marked resemblance to those of the pomegranate, and the bulbs were tested on sheep and rabbits, and were found to be extremely poisonous. In rabbits 50 grams of the bulb caused symptoms to start within three minutes after dosage. The animals showed marked restlessness, pronounced dyspnoea, and an accelerated pulse. After a few minutes, signs of paralysis appeared, the animals being unable to keep the head up. Furthermore, the pulse became imperceptible, the animals dying within twelve minutes after dosage under symptoms of asphyxia. These symptoms and the post-mortem appearances seem to point to an acute heart poison. 25 grams of bulbs caused a marked diarrhoea, in addition to the above symptoms, the animals dying in a state of complete paralysis within twenty-four hours after dosage.

Sheep dosed with 200 grams of bulbs showed the same train of symptoms. The ripe seeds proved to be about half as toxic as the bulbs.

Post-mortem Appearances.—Marked general cyanosis, pronounced dilation of the heart, both ventricles being distended with coagulated blood masses; and marked hyperaemia of the lungs. In the more protracted cases there was an acute haemorrhagic gastritis.

CRINUM SPECIES, PROBABLY *C. LONGIFOLIUM* THUNB.

Synonyms.—Seeroogblom, Oranje Rivier lelie.

Origin.—C. W. du Toit, Slangfontein, Heidelberg, Transvaal.

Two rabbits, drenched with 10 and 60 grams of bulbs and leaves respectively, showed no symptoms of illness.

CACTACEAE.

OPUNTIA DECUMANA, VAR. *SPINOSA*.

Synonyms.—Turksvy, prickly pear.

History.—Miss K. Jacobs, Platberg, Content Rail, via Kimberley, forwarded ten muscovi ducks to Onderstepoort on the 27/9/27 suffering from lameness. She had been mixing cut-up prickly-pear leaves with the food of these birds, and suspected these leaves to be the cause of the trouble.

Feeding-experiments with prickly pear growing at Onderstepoort were conducted with four muscovi ducks. As these birds refused to take the minced leaves, either as such or mixed with other foodstuffs, they had to be forced-fed. Each of them received 480 grams during a period of twelve days at the rate of 40 grams daily. No symptoms developed up to two months after the discontinuation of the experiment.

CAESALPINEAE.

ELEPHANTORRHIZA BURCHELLI, BENTH.

Synonyms.—Elandsboontjie, looiersbossie.

History.—This is the first toxic record of this plant. The beans were forwarded by Dr. Phillips, of the Division of Botany, Pretoria, with the remark that they are suspected of having caused death in a native.

Toxic Principle.—The beans contain a strong irritating principle. When chewed, they first have a sweetish taste and then cause feeling of burning. The toxin(s) is soluble in water and insoluble in absolute alcohol and ether. On extracting the ground beans with ether, a non-toxic, thick, yellowish, tasteless oil is obtained. These beans contain 10 per cent. by weight of this oil. After the clarification of the aqueous extract with basic lead acetate, removing the lead with a saturated solution of sodium carbonate, and evaporating the filtrate, a thick, light brown, resinous substance is obtained, which proved to be very toxic to guinea-pigs when injected subcutaneously.

Symptoms.—0.75 gram equivalent of the aqueous extract of the beans injected subcutaneously into guinea-pigs caused extensive necrosis of the subcutaneous tissues at the point of injection, in addition to an acute catarrhal gastro-enteritis and oedema of the lungs. The M.L.D. for rabbits per os varied from 5 to 7½ grams per kilogram body weight. These animals showed marked apathy, inappetence, and a profuse foetid diarrhoea, dying within twenty-four hours after dosage in a state of complete exhaustion.

In sheep 250 grams caused death under symptoms of complete exhaustion, within twenty-four hours, after having shown apathy, general weakness, an accelerated pulse and respiration, marked abdominal pain, and diarrhoea.

Post-mortem Appearances.—A pronounced acute catarrhal gastro-enteritis with numerous haemorrhages in the gastric and intestinal mucosa; marked degeneration of the liver.

CARICACEAE.

CARICA PAPAYA.

Synonym.—Pawpaw.

History.—The following is an extract from an answer to query No. 9380, which appeared in the *Farmers' Weekly* of the 4th July, 1928:—" Well, if you take two or three pawpaw leaves, stems as well, and mince them through a mincing machine, or chaff-cutter, very fine, and mix it with a little bran, and give it to your horse after starving it for a day, you will find that it passes all the bots in a few hours. If you like, you can repeat the dose in a couple of days' time."

Before experimenting on horses, it was ascertained whether the leaves have no toxic properties. For this purpose each of two rabbits was drenched with a 100 grams of the leaves. Both animals remained healthy. Four horses were then treated as prescribed in the above letter, three receiving 250 grams of the pawpaw leaves (3 leaves), and the remaining one 400 grams (5 leaves). None of these horses passed any bots in the course of the week following the treatment. It was decided to kill one of the horses for post-mortem purposes, but owing to a misunderstanding during my absence from Onderstepoort, this decision was not executed. There is, however, no doubt that these horses were infected with bots, as amongst the many hundreds of horses which have been post-mortemed at Onderstepoort, none were found to be bot-free.

CHENOPODIACEAE.

SALSOLA KALI, Meyer.

Synonyms.—Russian thistle, saltwort, tumble-weed.

Origin.—Mr. Bouwer, Mayfield, Albany.

History.—This plant was suspected of having caused deaths amongst cows which were fed on lucerne mixed with saltwort. The sharp spines may pierce the skin and form festering sores.

The only material available was 400 grams of the half-dried plant in the flowering stage, and a sheep drenched with this quantity showed no symptoms of ill-health.

SUAEDA FRUTICOSA, Forsk.

Synonym.—Inkbos.

Origin.—J. van Zyl, Vanzylsdamme, Ladismith, C.P.

The quantity was too small to allow of any drenching experiments being carried out. Farmers maintain that sheep not accustomed to this plant will partake of it. It is said to produce a persistent black diarrhoea and death.

COMPOSITAE.

DIMORPHOTHECA CALENDULACEA, Harv.

Synonym.—Namaqualand daisy.

Origin.—Garden at Onderstepoort Students' Hostel.

Drenching experiments with sheep were carried out both with the pre-flowering and flowering stage of the above plant. These animals received quantities up to 500 grams in a single dose, without any deleterious effects. The picrate paper test, however, revealed the presence of a fair amount of hydrocyanic acid in all parts of the plant at all times of the day.

DIMORPHOTHECA SPECTABILIS.

Synonym.—Bietou.

Origin.—Old mealie lands at Davel and near Olifantsfontein Station, on the Pretoria-Johannesburg line.

Toxicity.—Drenching experiments showed that this plant is equally toxic in its different stages of development, and that there is no decrease in its toxicity whether sun or air dried. It was also ascertained that the plant was equally toxic at all times of the day. 25 grams of the freshly picked plant had no effect on sheep, whereas 100 grams caused transitory symptoms of dyspnoea, frothing at the nostrils and mouth, with complete recovery after seven to twelve hours. 130 grams caused the typical symptoms of hydrocyanic acid poisoning, to start within five minutes, and death within fifty-five minutes after dosage. The M.L.D. for sheep, therefore, lies between 100 and 130 grams of the fresh plant. When minced, this plant emits a strong smell of prussic acid, and after a while one feels difficulty in breathing.

Inhalation Experiment.—Fifty grams of the fresh plant were minced and placed in a crucible, which was put under a bell jar with two guinea-pigs. These animals showed lachrymation and symptoms of restlessness and dyspnoea within two minutes after enclosure in the bell jar. After ten minutes they were completely paralysed, and the respiration steadily decreased in frequency and depth, until death within twenty minutes after the commencement of the experiment.

Symptoms.—As the symptoms and post-mortem appearances of bietou poisoning completely coincide with the effects of prussic acid, it is superfluous to describe these in detail in all the animals—namely, guinea-pigs, rabbits, sheep, horses, and cattle—employed in these experiments. A horse drenched with 200 grams of the dried bietou (equivalent to 1 kilogram of the fresh plant) developed the following symptoms within half an hour: continual yawning, a reddish and subsequently a bluish discolouration of the visible mucous membranes, an accelerated and weak pulse, muscular tremors at different parts of the body, especially affecting the pectoral and triceps muscles, general weakness, staggering about, clonic spasms, and a very pronounced dyspnoea. Three hours after dosage its condition improved, and after a further two hours only a general weakness was present. Complete recovery took place within a further eight hours. A beast drenched with 250 grams of dried bietou (equivalent to 1,200 grams of the fresh plant) showed within five minutes after dosage, in addition to the symptoms described in the horse, opisthotonos, hoven, and great distress.*

ANTIDOTAL EXPERIMENTS.

Before describing these experiments, it is essential to say a few words about "geilsiekte." There is no doubt that this term is employed for quite a number of different diseases in sheep, especially in cases where sudden death occurs. Acute plant poisoning is very frequently called "geilsiekte" by farmers. In the drier parts of South Africa (Karroo), where spasmodic rains with a subsequent luxurious growth of grass occur, and where the blazing sun very frequently causes the wilting of the green grass, the term "geilsiekte" is applied almost without exception to prussic acid poisoning, caused by the ingestion of wilted grass. Experienced farmers maintain that the Government Wireworm Remedy and Cooper's Powder are effective preventives of "geilsiekte." In order to test this contention of the farmers, the undermentioned experiments were conducted with the following drugs: sulphur, partly dehydrated $CuSO_4$, as used in the Government Wireworm Remedy, sodium arsenite, Government Wireworm Remedy, and Cooper's Powder, which has a total sulphur-content of 68 per cent.

A.—PROPHYLACTIC TREATMENT.

Dried bietou was utilized as the source of hydrocyanic acid, the M.L.D. of sheep varied from 23 to 24 grams. Sheep were employed in these experiments, and were treated as folows:

(*a*) *Sulphur.*

A sheep received 2 grams of the ordinary commercial sulphur daily, for a period of seven days, and on the eighth day it was dosed with 26 grams of dried bietou

Results.—The animal showed very slight symptoms of dyspnoea, but recovered within a few hours.

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* Death occurred within half an hour after dosage.

(*b*) *Partly dehydrated* $CuSO_4$ (*ingredient of Government Wireworm Remedy*).

A sheep received 0.5 gram of above drug daily for a period of seven days, and was drenched with 26 grams of bietou on the eighth day.

Result.—Died within one and a half hours after dosage.

(*c*) *Sodium arsenite* (*ingredient of Government Wireworm Remedy*).

A sheep received 0.125 gram of above drug daily for a period of seven days, and was dosed with 26 grams of bietou on the eighth day.

Result.—Died within three hours after dosage.

(*d*) *Government Wireworm Remedy.*

A sheep received 0.625 gram of above drug daily for a period of seven days and was drenched with 26 grams of bietou on the eighth day.

Result.—Died within 45 minutes after dosage.

(*e*) *Cooper's Powder.*

A sheep, dosed with 0.5 gram of the above drug daily for a period of seven days, died from arsenical poisoning during the night of the seventh day, so that the antidotal treatment could not be carried out.

(*f*) *Control sheep dosed with* 26 *grams of bietou died within quarter of an hour.*

Conclusion.

Prophylactically sulphur seems to have a beneficial effect on prussic acid poisoning, provided the dose of the latter is not too big and the correct dose of sulphur is given. Chemically this action of sulphur on hydrocyanic acid is easily explicable, namely, the innocuous compound sulphocyanic acid (HCNS) being formed.

B.—SIMULTANEOUS TREATMENT.

In the following experiments the dry bietou and the drugs were mixed immediately before the sheep were drenched.

(*a*) *Sulphur.*

A sheep received 26 grams of bietou and 10 grams of sulphur.

Result.—Remained healthy.

(*b*) *Partly dehydrated* $CuSO_4$ (*as used in Government Wireworm Remedy*).

A sheep received 26 grams of bietou and 10 grams of above drug.

Result.—Animal showed slight symptoms of dyspnoea and recovered within two hours.

(*c*) *Sodium arsenite* (*used in Government Wireworm Remedy*).

A sheep was drenched with 26 grams of bietou and 0.125 gram of above drug.

Result.—Animal died within one and a half hours after dosage.

(*d*) *Government Wireworm Remedy.*

A sheep was drenched with 26 grams of bietou and 0.625 gram of above drug.

Result.—Animal showed slight symptoms of dyspnoea, but recovered within a few hours. It will be noted that in Experiment (*a*) the sheep which received the Government Wireworm Remedy daily for a period of seven days did not survive the same dose of bietou.

(*e*) *Cooper's Powder.*

A sheep was drenched with 26 grams of bietou and 2 grams of above drug.

Result.—Animal showed slight symptoms of dyspnoea, but recovered.

(*f*) The control sheep dosed with 26 grams of bietou died within a quarter of an hour.

Conclusion.

Sodium arsenite does not prevent hydrocyanic acid poisoning, whereas in this experiment, at least, there is a distinct antidotal action in the case of sulphur, $CuSO_4$, the Government Wireworm Remedy, and Cooper's Powder, that is, all the drugs containing sulphur.

These experiments, as well as the effects of continual dosing with sulphur, are to be repeated on a larger scale.

It was furthermore attempted to produce a chronic case of prussic acid poisoning, and for this purpose Sheep No. 18933 was dosed as follows:—

On 12/6/28 : 10 grams of dried bietou;
13/6/28 : 10 grams of dried bietou;
14/6/28 : 10 grams of dried bietou;
15/6/28 : 10 grams of dried bietou;
16/6/28 : 10 grams of dried bietou;
18/6/28 : 10 grams of dried bietou;
19/6/28 : 10 grams of dried bietou;
20/6/28 : 15 grams of dried bietou;
21/6/28 : 20 grams of dried bietou, showed symptoms of a stiff gait, but recovered;
22/6/28 : 20 grams of bietou: dyspnoea and stiff gait;
23/6/28 : 20 grams dried bietou: dyspnoea and stiff gait;
24/6/28 : 20 grams of dried bietou: dyspnoea and stiff gait;
25/6/28 : 25 grams of dried bietou: died within three hours.

The microscopical examination of the organs of the sheep which died from bietou poisoning revealed haemorrhages in the lymph glands and a marked congestion of the spleen.

As it is essential to know how long sulphur, after it had been dosed to a sheep, will protect it from moderate doses of prussic acid, the following experiment was conducted: A sheep was dosed with 15 grams of sulphur and received 28 grams of dried bietou the following day, without any deleterious effects. When this dose of bietou was repeated on the second day after the sulphur had been administered, the animal succumbed to prussic acid poisoning.

This point is of importance in the combating of "geilsiekte" (wilted grass form), as far as licks are concerned. From the above experiment it is evident that the animals should receive small quantities of sulphur, at least three times a week, in order to prevent "geilsiekte." As it is a general practice amongst farmers in South Africa to allow sheep continual access to different kinds of licks, it is most practicable to mix the sulphur ($\frac{1}{2}$ lb. per 100 sheep) with these licks in times when "geilsiekte" is liable to occur.

Toxic Principle.—All the usual methods of isolation of the cyanogenetic glucosides were employed in the attempts to isolate the toxic principle of *Dimorphotheca spectabilis.* No results were obtained, as in each case the evaporated hot and cold extracts proved to be non-poisonous on subcutaneous injection into guinea-pigs.

Most probably the hydrocyanic acid is present in the leaves in the form of a very unstable cyanogenetic glucoside, as the process of drying the leaves in the sun has no effect on the toxicity of the leaves, even after months of storage in an open vessel. Had the prussic acid been present as such, this would not have been the case.

DIMORPHOTHECA ZEYHERI, SAND.

Synonym.—Jakhalsbos.

Origin.—G. Whitehead, P.O. Meadows, Bloemfontein.

The picarte paper test revealed the presence of a large amount of prussic acid.

A sheep, drenched with 150 grams of the plant in the pre-flowering stage, developed typical symptoms of prussic acid poisoning within three minutes, and died within half an hour after dosage.

EPALTES ELATA STEETZ.

Origin.—Mr. Meyer, Rietfontein, Bloemhof.

A sheep received a kilogram of this plant in the flowering stage on two successive days, without any deleterious effects.

GEIGERIA SPECIES.

Vermeersiekte (Misbeksiekte).

Introduction.—A full account of "vermeersiekte" caused by *Geigeria* "passerinoides" is given by Du Toit in the 13th and 14th Reports of the Director of Veterinary Education and Research, pp. 107-153.

The information given below is based both on field observations and experiments carried out at the Onderstepoort laboratories. Recently the following *Geigeria* species were proved toxic:

1. *Geigeria aspera.*
2. *Geigeria zeyheri.*
3. *Geigeria* species, National Herbarium No. 7891. This specimen subsequently proved to be *Geigeria aspera.*

Origin.—(*a*) *Geigeria aspera*: (1) T. Ninham, Boschkoppies, Kroonstad, O.F.S.; (2) Mr. De Witt, Edenville, O.F.S., Railway Premises, Devon, Tvl.; (3) D. Sevenster, Dankbaar, Senekal, O.F.S. (*b*) *Geigeria zeyheri*: Along the Railway line, Pretoria North. (*c*) *Geigeria* sp. Nat. Herb. 7891: Mr. Rautenbach, Potberg, Standerton.

Toxicity.—There is a general belief amongst farmers that the small white worm in the flowers of these plants is the cause of "vermeerziekte." This, however, is not the case, as the pre-flowering stage is even more toxic than the flowering stage; the toxicity of *Geigeria aspera* decreases on storage until complete disappearance after a month to six weeks.

As far as our present knowledge concerning the *Geigeria* species goes, *Geigeria aspera* is about three times as toxic as *Geigeria zeyheri*, and about twelve times as toxic as *Geigeria* passerinoides. Three hundred grams of *Geigeria aspera* in the pre-flowering stage causes death in a sheep within 40 hours after dosage, after the animal had developed the typical symptoms of "vermeersiekte."

Another point of interest is the seasonal variation in the toxicity of *Geigeria zeyheri*. The tests, which were carried out at intervals of 14 days, covered a period of 14 months (December, 1927, to end of January, 1928).

This plant was found to be very toxic to sheep during December, 1927, and January, 1928. Towards the end of January heavy rains fell at Onderstepoort, and the toxicity of the plant was found to have disappeared from the middle of February, 1928. It was found impossible to kill sheep during the period February to September, 1928, with *Geigeria zeyheri* growing along the Railway line at Pretoria North. From the beginning of October, 1928, 900 grams (the usual lethal dose) again killed sheep.

Toxic Principle(s).—Various attempts employing the usual methods of isolation have met with no success.

Symptoms.—According to observations in the field and experimental results at Onderstepoort, it is proposed to divide "vermeersiekte" into four different forms, namely: (*a*) the vomiting form, (*b*) the form in which bloating is the outstanding feature, (*c*) the stiff form, and (*d*) the paralytic form. An animal may, of course, suffer from one or more of the above forms. Boer goats seem to be more susceptible to the paralytic form than sheep. One often finds these animals lying down and unable to rise on account of paralysis of the hindquarters.

It may happen that sheep and goats die suddenly after the ingestion of big quantities of the "vermeerbossies," but the usual course taken by the disease is chronic vomiting and diarrhoea caused by the constant irritation of the gastro-intestinal tract. The affected animals lose in condition and ultimately die from exhaustion.

Dogs and pigs could not be killed with *Geigeria aspera*, as they vomited within five minutes after dosage, thus expelling the plant before the toxin could be absorbed.

Microscopical Pathological Anatomical Diagnosis.—Fatty changes in the liver and myocard, and a marked haemorrhagic lymphadenitis.

GNAPHALIUM LUTEO-ALBUM L.

Synonyms.—Roerkruid, Cudweed.

Origin.—Government Veterinary Officer, Kokstad.

50 grams of the plant in the flowering stage had no effect on rabbits.

HELICHRYSUM SPECIES, PROBABLY *H. KRAUSSII.*

Origin.—C. E. Jones, Tarporley, P.O. Baroda, Cradock.

350 grams of the dry plant in the flowering stage had no effect on a sheep.

INULA GRAVEOLENS L.

Synonyms.—Khaki-bush. Khaki-weed.

Origin.—Wellington.

A rabbit, dosed with 50 grams of the dry plant in the flowering stage, died on the sixth day after dosage from pneumonia and pleuritis.

NIDORELLA ANOMALA STEETZ.

Origin.—Mr. Wylie, Sonderwater, Premier Mine, Pretoria.

500 grams of the fresh plant in the flowering stage had no deleterious effects on a sheep.

NIDORELLA RESEDAEFOLIA D.C.

Origin.—C. E. Jones, Tarporley, P.O. Baroda, Cradock.

Only two small specimens of this plant were sent in so that no tests could be carried out. Farmers hold the opinion that it causes staggers in horses.

PENTZIA GLOBIFERA.

Synonym.—Stinkkruid.

Origin.—J. M. Dames, Leeuwfontein, Murraysburg, Cape.

This plant is suspected of causing enzootic icterus in sheep, but no symptoms developed in sheep drenched with 300 grams of the dry plant in the flowering stage.

PTERONIA PALLENS L.F.

Synonyms.—Witgatbossie, witbossie, scholtzbossie, aasvoëlbos.

Origin.—(1) W. A. O'Shea, Senior Sheep Inspector, Calvinia, C.P.; (2) J. van Zyl, Vanzylsdamme, Ladismith, C.P.

It frequently happens that stock, newly introduced into areas where this plant grows, partake of it with fatal results.

Henning fed this plant with fatal results to sheep at Grahamstown. At Onderstepoort a sheep, drenched with 300 grams of the above dry plant in the flowering stage, died suddenly after 30 hours without having shown any conspicuous symptoms.

Post-mortem Appearances.—Marked general cyanosis; hydroperitoneum and hydrothorax, the fluid having an intense yellowish colour; numerous subendocardial haemorrhages in left ventricle; slight hyperaemia of lungs; marked venous hyperaemia and degeneration of liver; oedematous swelling of periportal, retropharyngeal, and submaxillary lymph glands; haemorrhages; haemonchosis.

SCHISTOSTOPHIUM CRATAEGIFOLIUM FENZL.

Origin.—Mr. Wylie, Sonderwater, Premier Mine, Pretoria.

200 grams of the above plant in the flowering stage had no effect on sheep.

SENECIO ANGUSTIFOLIA, WILLD.

Origin.—C. E. Jones, Tarporley, P.O. Baroda, Cradock.

Only two small specimens were forwarded to Onderstepoort, with the remark that the plant is suspected of causing staggers in horses.

SENECIO FREMONTI.

This plant, according to Dr. L. van Es, University of Nebraska, College of Agriculture, Lincoln, causes a disease in horses closely corresponding to "dunsiekte" in South Africa. Feeding experiments were carried out at the above University.

SENECIO LATIFOLIUS D.C.

Synonyms.—Dou's cabbage, Molteno-disease plant.

Origin.—J. C. Mossop, Cliffdale, Hlobane, Vryheid, Natal.

Toxic Principle.—H. E. Watt (1909) isolated two alkaloids, senecifoline and senecifolidine, from the above plant.

Symptoms.

Sheep.—A sheep, receiving 5 grams of dry ground Senecio daily, showed a continuous loss in condition until death on the sixteenth day after the commencement of the experiment. The total quantity of plant drenched was 80 grams.

Another sheep, receiving 10 grams daily, died on the eleventh day after a total dosage of 111 grams of dry Senecio. The only symptom noticed was dyspnoea. The M.L.D. of the fresh plant for sheep is approximately 450 grams.

Post-mortem Appearances in Sheep.—The following leisons were present:—General cyanosis, slight general icterus, marked hydroperitoneum, hydrothorax, subepicardial and subendocardial haemorrhages, oedema and hyperaemia of the lungs, a marked acute parenchymatous hepatitis with pronounced pigmentation of the liver, a pronounced acute catarrhal cholecystitis, a pronounced gelatinous infiltration of the folds of the abomasum, an acute catarrhal duodenitis, haemorrhages in retropharyngeal and submaxillary lymph glands, oedematous swelling of the bronchial and mediastinal lymph glands.

Horses.—A horse was drenched as follows:—

12/11/28: 300 grams of dry *Senecio latifolius.*
14/11/28: 100 grams of dry *Senecio latifolius.*
15/11/28: 250 grams of dry *Senecio latifolius.*

Animal losing in condition, showing slight icterus and inappetence. In the course of the next three days the icterus became more intense.

19/11/28: 250 grams of Senecio.

Animal very pathetic, standing with the head in the corner of the box and with closed eyes. Signs of colic developed, pulse weak but not accelerated, respiration laboured, intense icterus.

20/11/28: Animal died at 1 a.m. under symptoms of complete unconsciousness.

Post-mortem Appearances.—A pronounced general icterus, marked hydroperitoneum, subepicardial and subendocardial haemorrhages, a marked acute parenchymatous hepatitis with necrosis and partial cirrhosis, pancreas showed haemorrhages and a marked yellowish gelatinous infiltration, haemoglobinuria, acute catarrhal gastro-enteritis with blood transfusion into lumen of stomach and colon, pronounced gelatinous infiltration of submucosa of caecum and perirectal tissues.

Cattle.—A one-year-old beast received the following quantities of *Senecio latifolius*:—

7/11/28: 30 grams.
8/11/28: 30 grams.
9/11/28: 30 grams.
12/11/28: 250 grams.

13/11/28: Inappetence, apathetic, lying down and unwilling to rise; when urged, it rose leaning against wall of box, frequent straining without passing any faeces, pulse 128, respiration 80.

14/11/28: Stable floor covered with blood and animal was still bleeding from left nostril, frequent straining, points of horns showed evidence of pushing, temperature 102.3° F., animal made attempts to rise but was unable, groaning. Animal died at 1.30 p.m.

Post-mortem Appearances.—Anaemia; multiple haemorrhages in subserosal, subpleural, intermuscular, peritrachael, perioesophageal, and subcutaneous tissues; acute parenchymatous hepatitis with partial cirrhosis; a very marked acute catarrhal cholecystitis with extravasation into the wall, which showed a pronounced infiltration with a yellowish gelatinous substance; gall mixed with blood; tumor splenis, with numerous subcapsular haemorrhages; impaction of omasum; gelatinous oedema of folds of abomasum with haemorrhages in mucosa; acute catarrhal duodenitis and jejunitis; blood transfusions into lumen of caecum and colon; perirectal tissues markedly infiltrated with a yellowish gelatinous material.

SENECIO PUBIGERUS, LINN.

Origin.—Mr. Parkin, Lecturer in Veterinary Science, Elsenburg Agricultural College.

Only one specimen was received at Onderstepoort, with the remark that this plant is suspected of causing cirrhosis of the liver in mules.

SENECIO VENOSUS.

Origin.—(1) Corner of Lawley and Schoeman Streets, Pretoria; (2) Hammanskraal.

Two sheep, receiving 200 grams and a kilogram respectively of the fresh plant in the flowering stage, developed no symptoms; also a rabbit received 30 grams with negative results.

TAGETES MINUTA L.

Synonyms.—Khakibush, Mexican marigold.

Origin.—Mr. Hains, Union Farm, Pyramids.

A sheep received 700 grams of the fresh plant in the flowering stage, with negative result.

CRASSULACEAE.

COTYLEDON ORBICULATA L.

Synonyms.—Honde-oor, vark-oor, pig's ear, kouterie.

Origin.—(1) Magaliesberg, Pretoria North; (2) P. L. le Roux, Pokkraal, Worcester; (3) L. J. Matthee, Zeekoeriver, Humansdorp.

Symptoms.—These were described by Henning in the 11th and 12th Reports of the Director of Veterinary Education and Research.

The specimens from Worcester and Humansdorp proved to be toxic to sheep and goats.

Toxicity.—Comparative tests of the toxicity of *Cotyledon orbiculata*, growing in the Magaliesberg and in the poison garden at Onderstepoort, showed that the former specimens were three times as toxic as those growing in the heavy clay soil in the poison garden. This formed the basis of the following experiment. A specimen was brought from the Magaliesberg, and the subcutaneous M.L.D. per 500 gram guinea--pig ascertained. This specimen was then cut in half, one of which was planted in sandy soil and the other in heavy clay soil in the poison garden. They were watered once a week. Five and a half months after the transplantation they were retested, and it was found that the sandy soil part of the plant had lost halt of its toxicity, whereas the clay soil part had doubled its toxicity. On mincing the two plants, it was, however, found that the sandy soil specimen had a greater water-content than the clay soil specimen.

It was ascertained that the young leaves, middle-sized leaves, and old leaves are approximately equally toxic and that the flowers are twice as toxic as the leaves; and, furthermore, that the process of slicing and desiccation in the sun has no effect on the toxicity of the leaves.

COTYLEDON PAPILLARIS.

Origin.—Grahamstown.

A sheep received 900 grams of above plant (buds, flowers, and leaves) in the flowering stage without any deleterious effects.

COTYLEDON VENTRICOSA Burm.

Synonym.—Nenta.

Origin.—J. W. van Rensburg, Scheepersdrift, Uniondale Road, Cape.

The above specimen proved to be extremely toxic on subcutaneous injection of the evaporated 96 per cent. alcoholic extract into guinea--pigs.

EBENACEAE.

ROYENA LUCIDA L.

Synonyms.—Swartbas, blackbark, wild coffee

Origin.—J. Laing, Blackwoods, Seymour, C.P.

This plant is eaten by sheep during the winter months, or dry seasons, and is suspected of being poisonous. A sheep, drenched with 200 grams of the dry leaves and berries, remained completely healthy.

EUPHORBIACEAE.

EUPHORBIA ELLIPTICA Thb.

Origin.—L. J. Matthee, Zeekoeriver, Humansdorp.

The quantity forwarded was too small to allow of any test being carried out. It is suspected of having caused deaths in sheep.

EUPHORBIA SPECIES probably *E. MURICATA* Thb.

Origin.—L. J. Matthee, Zeekoeriver, Humansdorp.

300 grams of the fresh plant had no effect on a sheep.

EUPHORBIA PUBESCENS.

Origin.—George.

The Government Veterinary Officer reports that the above plant causes constipation and narcosis when eaten by stock not accustomed to it, but feeding experiments carried out at the Allerton Veterinary Laboratory, Pietermaritzburg, from the 7th-11th July, 1924, were negative.

EUPHORBIA PULVINATA Marl.

Synonyms.—Voetangel, pincushion.

Origin.—L. Moreland, Redstones, Klip River, Natal.

Five hundred grams of the fresh plant had no effects on a sheep.

EUPHORBIA RHOMBIFOLIA.

Origin.—J. Adams, Warrenton.

Only a small specimen was forwarded. Farmers in the Carnarvon and Williston districts maintain that this plant causes urinary trouble in goats.

EUPHORBIA TRUNCATA.

Synonym.—Vingerpol.

Origin.—Mr. Van Niekerk, Vogelfontein, Bethlehem, O.F.S.

Only a small specimen of this plant was forwarded, with the remark that it is suspected to have caused deaths in lambs.

RICINUS COMMUNIS L.

Synonym—Castor-oil plant.

Origin.—Onderstepoort poison garden.

A rabbit, dosed with 45 grams of the green fruit in its youngest stage of development, showed a profuse diarrhoea within two hours, and died in a state of complete exhaustion within seven hours after dosage.

Post-mortem Appearances.—An acute haemorrhagic gastro-enteritis; heart markedly dilated and filled with coagulated blood masses.

GRAMINAE.

AVENA SATIVA

(Moulded Oats.)

Two hundred grams of badly moulded oats were drenched to sheep with negative results.

CYNODON BRADLEYI.

Specimens of this grass growing on lawns at Onderstepoort were found to contain large amounts of prussic acid, even as early as 6.30 in the morning in mid-summer. Rabbits dosed with quantities up to 100 grams of this grass showed no symptoms of poisoning, due to the fact that most of the prussic acid escaped during the process of mincing.

LOLIUM TEMULENTUM L.

Synonyms.—Drabok, cheat, bearded darrel.

Origin.—(1) Riversdale; (2) M. Kennedy, Drew, C.P.; (3) P. L. le Roux, Pokkraal, Worcester.

Quite frequently farmers have suspected these seeds to have caused symptoms and death in horses.

Rabbits are exceedingly fond of these seeds, and have ingested as much as 200 grams per day, without any deleterious effects.

The specimens tested, however, consisted of healthy seeds, whereas it is held that only fungus-infested seeds cause symptoms of poisoning and death. The fungus is known as *Endoconidium temulentum*, and the toxic principle as picrotoxin.

HALORRHAGIDACEAE.

GUNNERA PERPENSA, LINN.

Synonym.—Wilde ramenas.

Origin.—A. G. Robertson, Maquabie, Amersfoort, Transvaal.

Extractions of the roots of this plant are used by farmers and natives to expel the retained afterbirth.

A sheep drenched with 700 grams of the fresh roots developed no symptoms of poisoning.

IRIDACEAE.

HOMERIA PALLIDA, BAKER.

Synonyms.—Transvaal yellow tulip, Transvaal geel tulp.

Origin.—(1) L. . Matthee, Zeekoeriver, Humansdorp; (2) Malcolm Thorrold, Coalbrook Siding, Ermelo.

In sheep 700 grams of this tulip in the flowering stage caused typical tulip symptoms and death within two hours after dosage.

MORAEA POLYSTACHYA, KER.

Synonyms.—Cape blue tulip, Kaapse blou tulp.

Origin.—Loerie Station, near Humansdorp.

Two hundred and fifty grams in the flowering stage caused typical tulip symptoms, and death in sheep within ten hours after drenching.

MORAEA SETACEA, KER.

Synonyms.—Blou tulp, blue tulip.

Origin.—Grahamstown.

Rabbits, dosed with quantities up to 40 grams of the above plant in the flowering stage, developed no symptoms of poisoning.

MORAEA SPATHACEA, KER.

Synonyms.—Nokha, Cape blue tulip.

Origin.—(1) Jeffrey's Bay Station; (2) Queenstown.

Four hundred grams of the dry plant in the flowering stage killed a sheep within ten hours, whereas 80 grams killed rabbits within 1¼ hours after dosage.

LEGUMINOSAE.

CANAVALIA ENSIFORMIS.

The green beans of the above plant were sent in to Onderstepoort, with the request to test their effects on animals. Rabbits ingested up to 400 grams of the green husks and beans mixed with bread within a period of four days, without any deleterious effects.

A beast ingested 3½ lb. of the beans and husks during a period of six days, without developing any symptoms of ill-health.

CASSIA OBOVATA, COLLAD.

Synonym.—Wildsenna.

Origin.—Mr. Meyer, Rietfontein, Bloemhof.

Previous feeding tests proved this plant to be poisonous to stock, whereas 500 grams of the dry plant in the flowering stage drenched to sheep at Onderstepoort proved to be innocuous.

LANCHOCARPUS CAPASSA, ROLFE.

Synonym.—*Molana.*

Origin.—Lusaka, Northern Rhodesia.

Natives use this plant as a remedy for snake-bite. A rabbit dosed with 30 grams of the dry leaves developed no symptoms of poisoning.

LOTONONIS INVOLUCRATA, BENTH.

Origin.—P. L. le Roux, Pokkraal, Worcester.

In the fresh state this plant contains a large amount of prussic acid. 150 grams of the plant in a half-dry state caused marked symptoms of hydrocyanic acid poisoning and diarrhoea, the animal recovering after a period of three days. There is, however, no doubt that in the fresh state much smaller quantities of the plant will cause death in sheep, as during the process of desiccation a large amount of HCN is emitted.

STIZOLOBIUM DEERINGIANUM.

The green beans were forwarded by the Division of Botany, Pretoria, with the request to test their effects on animals.

The beans and husks were minced and mixed with bread and fed to two rabbits. They ingested 360 grams of the beans and husks during a period of four days, without any deleterious effects.

STIZOLOBIUM PRURIENS.

Synonym.—Velvet bean.

These beans were also forwarded by the Division of Botany with the above request.

During a period of 19 days a beast ingested 10½ lb. of beans and husks without any deleterious effects.

VIGNA VEXILLATA.

Synonym.—Cowpea.

Origin.—Boksburg.

This plant was suspected to having caused severe symptoms in calves.

A sheep, drenched with 400 grams of the fresh plant in the pre-flowering stage, developed no symptoms of ill-health.

LILIACEAE.

ASPARAGUS MEDEOLOIDES, Thunb.

Synonym.—Cape smilax.

Origin.—Humansdorp.

Four hundred grams of fresh plant in the flowering stage had no deleterious effect on a sheep.

BULBINE ASPHODELOIDES (L) Roem & Schutt.

Synonyms.—Wilde kapiva, geel kattestert.

Origin.—T. Ninham, Boschkoppies, Kroonstad.

A sheep, drenched with 400 grams of fresh roots, leaves, and flowers, developed no symptoms of ill-health.

ORNITHOGALUM AURANTIACUM.

Synonyms.—Yellow chinkerinchee.

Origin.—P. L. le Roux, Pokkraal, Worcester.

A rabbit dosed with 40 grams of the fresh plant in the flowering stage remained healthy.

ORNITHOGALUM CAUDATUM, Ait.

Origin.—L. J. Matthee, Zeekoeriver, Humansdorp.

A sheep received 700 grams of the fresh plant in the flowering stage on each of two successive days, without showing any symptoms of ill-health.

ORNITHOGALUM ECKLONII.

Origin.—(1) Grahamstown; (2) Port Elizabeth.

A rabbit, drenched with 35 grams of fresh leaves and bulbs, developed no symptoms of ill-health.

ORNITHOGALUM LACTEUM.

Synonym.—Chinkerinchee.

Origin.—P. L. le Roux, Pokkraal, Worcester.

Symptoms.—A sheep, drenched with 900 grams of the fresh plant in the flowering stage, developed symptoms of poisoning on the second day after dosage. It showed a profuse fluid diarrhoea, accelerated pulse and respiration, temperature 104.5° F. The animal staggered about in the box, frequently supporting itself against the wall. There was severe abdominal pain. Eventually the animal died in a state of complete exhaustion.

Post-mortem Appearances.—General cyanosis, subepicardial and subendocardial haemorrhages, marked hyperaemia and oedema of the lungs, pronounced hyperaemia of the spleen, an acute catarrhal gastro-enteritis.

SCILLA COOPERI, Hook.

Origin.—Heidelberg, Transvaal.

Rabbits, dosed with 75 grams, and sheep with 250 grams of the fresh leaves, flowers, and bulbs, developed no symptoms of poisoning.

SCILLA RIGIDIFOLIA, Kunth.

Synonym.—Wild squill.

Origin.—Bloemfontein.

Fifty grams of the fresh plant in the flowering stage had no effect on rabbits.

URGINEA BURKEI, Baker.

Synonym.—Transvaal slangkop.

Origin.—Grahamstown.

Symptoms.—Five hundred grams of the fresh bulb (plant being in the budding stage) caused death in sheep within 12 hours after drenching. The symptoms set in with laboured breathing, diuresis, and bloating. The condition grew serious within a few hours. The pulse became extremely accelerated and weak, cyanosis and a profuse fluid diarrhoea were present.

Post-mortem Symptoms.—General cyanosis, numerous haemorrhages in subcutaneous tissues; marked hyperaemia and oedema of the lungs with numerous haemorrhages in the submucosa of the trachea; marked haemorrhagic swelling of the retropharyngeal submaxillary, bronchial, and mediastinal lymph glands; and an acute catarrhal duodenitis and jejunitis.

LOGANIACEAE.

STRYCHNOS HENNINGSII, Gilg.

Synonyms.—Hardepeer, hard pear.

Origin.—Transkei.

Symptoms.—Rabbits dosed with 50 grams of the dried bark collected in August, 1928, developed typical symptoms of strychnine poisoning, and died within 15 minutes after dosage. The post-mortem was completely negative.

STRYCHNOS MITIS.

Origin.—Transkei.

Fifty grams of the dried bark had no effect on rabbits.

MELIACEAE.

MELIA AZEDARACH, Linn.

Synonyms.—China tree, Chinese umbrella tree, china berry, Pride of India, Sering.

Origin.—The experiments were carried out with berries collected from trees growing at Onderstepoort, although specimens from other parts of South Africa all proved to be toxic.

A full report on the toxicity of these berries is appearing in the next number of the *Transactions of the Royal Society of South Africa* (Steyn and Rindl).

The inconclusive nature of the recorded evidence formed the basis of these experiments.

Rabbits.

Symptoms.—The M.L.D. of the dry berries varied from 4 to 6 grams per kilogram body weight. Excitement and subsequent paralysis and diarrhoea were the most outstanding symptoms in these animals.

Post-mortem Appearances.—The blood coagulated very gradually and was of an intense dark red colour, and an acute catarrhal or haemorrhagic gastro-enteritis.

Birds.

Four muscovi ducks were force-fed daily with 30 grams of the fruit for three days, without developing any symptoms of poisoning.

Two fowls force-fed daily with 50 grams of the fruit for a period of three days developed symptoms from the third day after the commencement of the experiment. The birds were very apathetic and showed profuse greenish diarrhoea. In the course of two days they became very weak, tumbling about until they were unable to rise. It was apparent that they were paralysed before death, which occurred from six to eight days after the commencement of the experiment.

Post-mortem Appearances.—An acute catarrhal ingluvitis and gastro-enteritis.

Dogs.

As these berries were vomited almost immediately after dosage, it was impossible to kill dogs with the berries.

Sheep.

Eight hundred grams of the dry berries caused symptoms to start within three hours after dosage. The most outstanding symptoms were excitement, later on apathy, cyanosis, weak pulse, and laboured respiration. Death took place within 18 hours after dosage.

Post-mortem Appearances.—General cyanosis, blood intense dark red and coagulated very slowly; the subcutaneous blood-vessels, especially those of the front quarters, were markedly injected with blood; fatty degeneration of the liver; tumor splenis; a marked general swelling of all the lymph glands, and a pronounced acute catarrhal abomasitis and enteritis.

Goats.

Quantities up to 400 grams had no effect on goats, whereas 800 grams caused symptoms with recovery within three days. A kilogram of the dry drupes caused restlessness, clonic spasms of the diaphragm, great distress, dyspnoea, a weak and fat pulse, hoven and paralysis. As the paralysis, which was most marked in the hindquarters, persisted for several days, the animal was killed for post-mortem purposes.

Post-mortem Appearances.—Dilatation of the right ventricle, a bilateral purulent conjunctivitis, a fibrinous pleuritis, bronchopneumonia, oedema of the mucous membrane of the abomasum, Stilesia hepatica with cirrhosis of the liver, and an abnormal amount of fluid in the spinal canal.

Pigs.

The M.L.D. per 75-kilogram pig varied from 150 to 200 grams of dry berries.

In pigs the following symptoms appeared:—Restlessness, vomiting, dyspnoea, cyanosis, and a weak and accelerated pulse, the animals dying within $2\frac{1}{4}$ hours after dosage in a state of convulsions and suffocation.

Post-mortem Appearances.—Cyanosis; stomach markedly distended with gas, and hyperaemia of the mucosa; congestion of the liver.

Microscopical Pathological Anatomical Diagnosis.—Fatty degeneration and hyperaemia of the liver and kidneys, and degeneration of the sciatic nerve (goat).

SUMMARY.

1. Pigs, sheep, goats, rabbits and guinea-pigs are susceptible to syringa berry poisoning, pigs being most susceptible, and goats less so than sheep.
2. Muscovi ducks could not be killed with relatively high doses of berries, whereas fowls succumbed to doses of 150 grams, force-fed during a period of three days.
3. Dogs vomited before the poison could be absorbed and consequently showed no symptoms of poisoning.
4. The most outstanding symptoms produced by the berries are excitement, paralysis, and narcosis, death occurring through suffocation.
5. The toxin is seated in the epicarp only.
6. The toxin is thermostable and soluble in alcohol, ether, and chloroform. The toxin is not of the nature of alkaloids, toxalbumins, or glucosides, and most probably belongs to the indefinite group known as " bitter principles."

MELIANTHACEAE.

MELIANTHUS COMOSUS, Vahl.

Syonnyms.—Kriudjie-roer-my-niet, truitjie-roer-my-niet.

Origin.—B. J. LOOTS, Brewershoek, Britstown, C.P.

Symptoms.—A sheep drenched with 80 grams of dry leaves, flowers, and young fruits, developed symptoms of poisoning within three hours. The most outstanding symptoms were dyspnoea, cyanosis, hoven, and an accelerated and weak pulse; death occurring within four and a half hours after dosage.

Post-mortem Appearances.—Slight hydropericardium, pronounced hyperaemia and slight oedema of the lungs, subepicardial haemorrhages, hyperaemia of the liver and kidneys; subcutaneous blood-vessels markedly injected, general cyanosis, rumen markedly distended with gas, and a marked acute haemorrhagic duodenitis and jejunitis.

MELIANTHUS MAJOR, Linn.

Synonyms.—Kruidjie-roer-my-niet, truitjie-roer-my-niet.

Origin.—(1) Mr. Deschamp, Alve Rich, Humansdorp; (2) P. L. le Roux, Pokkraal, Worcester.

Symptoms.—A hundred grams of the fresh plant in the preflowering stage caused symptoms within three hours. The animal showed great distress, profuse urination, oedema of the lungs, laboured breathing and an accelerated pulse, and died during the following night. 400 grams of the fresh plant caused death within four hours. Storage causes no decrease in the toxicity of this plant, as it still proved to be toxic 16 months after collection.

Post-mortem Appearances.—Pronounced general cyanosis, injection of the subcutaneous blood-vessels, marked hyperaemia and slight oedema of the lungs; hyperaemia of the liver, marked haemorrhagic swelling of the bronchial, mediastinal, retropharyngeal, and submaxillary lymph glands; numerous haemorrhages in mucosa of abomasum; and an acute catarrhal enteritis.

Microscopical Pathological Anatomical Diagnosis:—Extensive fatty degeneration of liver, hyperaemia of liver and kidney, and a marked haemorrhagic lymphadenitis.

PASSIFLORACEAE.

ADENIA SPECIES, NATIONAL HERBARIUM No. 7611.

Origin.—Mayfern, Eastern Transvaal.

Symptoms.—Sixty grams of the bulb caused dyspnoea and paralysis within five minutes after drenching. Fibrillary contractions of all the muscles of the body were seen, these persisted even up to five minutes after death, which occurred within ten minutes after dosage.

Post-mortem Appearances.—General cyanosis, marked hyperaemia of the lungs, pronounced hyperaemia of the liver, and heart in diastole.

ADENIA GLAUCA.

Origin.—Growing in a tin on the veranda of the Chemistry Building, Onderstepoort.

The leaves were found to contain a large amount of hydrocyanic acid. One hundred grams of the fresh leaves dosed to rabbits had no effect on these animals. On mincing the leaves, a strong smell of prussic acid is emitted, and the negative results may be ascribed to the escape of a large amount of the prussic acid.

PORTULACACEAE.

ANACAMPSEROS TELETHIASTRUM D.C.

Origin.—P. L. le Roux, Pokkraal, Worcester.

A sheep, drenched with 250 grams of the fresh plant in the flowering stage, had no ill-effects.

RANUNCULACEAE.

RANUNCULUS PUBESCENS, Thunb.

Synonym.—Kankerblaar.

Origin.—(1) Veterinary Research Laboratory, Nooitgedacht, Ermelo; (2) Banks of the Aapies River, Onderstepoort.

Mr. Le Roux, Veterinary Research Officer, Onderstepoort, was the first to prove this plant toxic to sheep at Nooitgedacht, Ermelo. The undermentioned experiments at Onderstepoort confirmed his findings.

Symptoms.—Three hundred and thirty grams of the fresh plant in the pre-flowering stage caused restlessness, hoven, an accelerated pulse, diuresis, and frequent defaecation in a sheep, within half an hour after dosage. The animal seemed to suffer severe pain, as it groaned continually, and was very restless. The animal recovered after three days.

Another sheep, dosed with 200 grams of the fresh plant in the flowering stage, showed similar symptoms, and in addition developed a profuse fluid greenish diarrhoea. As after three days the animal was still suffering severe pain, and there being no improvement in its condition, it was killed for post-mortem purposes.

The post-mortem revealed a pronounced acute catarrhal enteritis with numerous haemorrhages in the mucosa.

SOLANACEAE.

PHYSALIS MINIMA, L.

Synonyms.—Wild gooseberry, kalkoengif.

Origin.—Old lands at Onderstepoort.

The assertion by farmers that the above plant causes abortion in pregnant ewes, formed the basis of these experiments.

Six healthy heavy pregnant ewes in good condition were engaged in the experiments. Each of these animals were drenched daily for a period of 17 days with 600 grams of the fresh plant in its different stages of development. The ripe berries were gathered from the ground and also tested. Each ewe received a total amount of 10 1/5 kilograms of the plant. All the ewes remained healthy and gave birth to healthy lambs after the normal period of pregnancy.

SOLANUM CAPSICASTRUM.

Origin.—Klerksdorp Town Lands.

A rabbit, drenched with 100 grams of the ripe red berries, showed no ill-effects.

THYMELEACEAE.

ARTHROSOLEN POLYCEPHALUS, Mey.

Synonym.—Januarie-bos.

Origin.—Phillipstown.

Farmers believe this plant to be poisonous. On grinding the dried plant it causes a marked feeling of burning in the nose and throat.

Rabbits dosed with quantities up to 60 grams of the dry plant in the flowering stage, and sheep with one kilogram at the rate of 200 grams daily, developed no symptoms of ill-health.

GNIDIA CAPITATA, L.F.

Origin.—Pretoria (Erasmus).

This plant was suspected of having caused deaths amongst cattle. Only a small quantity was forwarded and a rabbit drenched with 30 grams of the fresh flowering plant had no ill-effects.

PEDDIEA AFRICANA, HARV.

Synonyms.—Sterkbos "Gashuana" isifufufu.

Origin.—(1) Haenertsburg, Transvaal; (2) Mount Shannon, Deepdale, Impendhle District.

Mr. Robinson, of the Division of Veterinary Services, Onderstepoort, conducted feeding experiments with a bovine at the Allerton Veterinary Laboratory, with negative results.

A sheep, drenched with 100 grams of the dry plant in the flowering stage, developed no symptoms of poisoning.

Natives are said to use this plant medicinally and criminologically.

VERBENACEAE.

CLERODENDRON TRIPHYLLUM.

Origin.—(1) A. Adendorff, Kendal, Natal.

Thirty grams of the dry flowering plant had no ill-effects on rabbits.

ZYGOPHYLLACEAE.

ZYGOPHYLLUM MICROCARPUM, LICHST.

Synonyms.—Ou-ooibos, armoedsbos, sandrepuis.

Origin.—Frasersburg, Laingsburg, Williston, Carnarvon.

The undermentioned experiments were carried out with material received from Mr. H. P. van Niekerk, Bleskrantz, Carnarvon, C.P. This plant was suspected of having caused heavy mortality in sheep transported by train from Middelburg, Cape, to Carnarvon. There are no previous records of the toxicity of this plant.

Symptoms.—A rabbit dosed with 25 grams of the dry flowering plant was found in a semi-paralytic state the next morning. The animal furthermore showed torticollis, quivering of the tail, an accelerated pulse and respiration, convulsions, diarrhoea, and a weak cornea reflex. It was lying on its side unable to lift its head. On the fourth day after dosage the animal died in a state of complete paralysis.

Post-mortem Appearances.—Pronounced hyperaemia of the lungs, subendocardial haemorrhages in left ventricle, an acute catarrhal gastro-enteritis, marked degeneration of the liver and kidneys.

A sheep was drenched with 600 grams of the dry plant. The next morning it was found lying down in the box with the head resting on the ground, and when touched it suddenly jumped into the air and fell on the floor, being unable to support its body on the legs. It rose with difficulty and walked with a swaying gait, showing marked ataxy. The heart-beat was very irregular and accelerated. On the third day after dosage, diarrhoea set in and the animal lost rapidly in condition. Furthermore, a quivering of the muscles, inappetence, listlessness, and an accelerated respiration were present. The animal seemed to have lost all control over the voluntary muscles. Death occurred on the tenth day after dosage. No more material was available to continue the experiments.

Post-mortem Appearances.—Anaemia, emaciation, marked hyperaemia of the lungs, liver, spleen, and kidneys; degeneration of the liver; impaction of the rumen and reticulum; haemorrhagic abomasitis and enteritis; haemorrhagic swelling of the bronchial, mediastinal, and retropharyngeal lymph glands.

Microscopical Pathological Anatomical Diagnosis.—Extensive fatty changes in the liver, kidneys, and myocard, and pigmentation of the spleen.

In conclusion, I would like to express my indebtedness to Dr. Phillips and Mr. Mogg, of the Division of Botany, for valuable information and plant specimens received from them. I am also indebted to Mr. Le Roux, of our Division, for the many plants he has supplied me with, and to Dr. Quin, who supervised the poisonous plant experiments during my absence from Onderstepoort.

Dr. De Kock kindly obliged by having the sections prepared and examined in the Department of Pathology of the Onderstepoort Laboratories.

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*15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.*

Vlei Poisoning.

(VALLEY POISONING.)
(VELD POISONING.)

PART I.

By C. P. NESER, B.A., D.Sc., M.R.C.V.S., Research Officer,
Onderstepoort.

HISTORY.

In the past several stockowners have reported mortality in cattle during the months of September to December. At first it was thought to be due to a change in grazing, which is so frequently responsible for alimentary disturbances, particularly of the nature of a paresis of the fore-stomachs, or a diarrhoea provoked by young vegetation.

The history supplied by a certain owner was emphatically against the above ideas, because cases of the disease in question never occurred prior to the decimation of his herd by East Coast fever in 1918. On account of East Coast fever, this owner tried to augment his income by means of certain crops which could be raised in his locality. The agricultural experiment was not very successful, and for this reason the farmer decided to revert to cattle rearing. After this decision, mortalities from a hitherto unknown disease occurred during the spring and early summer months. It may be mentioned here that on account of the agricultural activities the vlei (valley) vegetation extended to a considerable distance beyond its normal boundaries on either side of the stream, as seen in the photo.

This history immediately suggested the possibility of poisonous plants being responsible for the mortality in cattle.

After several visits to the farm, no explanation could be offered for the deaths, excepting upon the assumption that some unknown poisonous plant (or its parasites or saprophytes) had numerically increased to such an extent that in ordinary grazing the gross intake was sufficient to account for poisoning.

It was therefore advised that experienced botanists should investigate this possibility. This advice was accepted by the Director, but no known South African poisonous plants (or pathogenic parasites or saprophytes) were reported to be present there; on the contrary, it was suggested and subsequently disproved that the animals died from lead-poisoning.

EXPERIMENTS.

After this, the investigator resorted to the methods adopted by Andrews in his investigations in connection with pushing disease, i.e. tethering animals on selected sites. Mogg assisted in selecting the tethering sites. A complete botanical survey of the locality where the experiments were undertaken is given by Mogg in Part II of this report.

The tethering experiments were undertaken to elucidate the following points:—

(1) Could lead-poisoning be responsible for the symptoms and deaths?

(2) To establish the boundaries of dangerous areas, if lead-poisoning could be excluded.

(3) To discover the responsible poisonous agent, if lead-poisoning could be excluded.

These tethering experiments proved beyond doubt that:—

(*a*) Lead-poisoning was not responsible for the mortality in the cattle;

(*b*) plants commonly supposed to be poisonous could not be incriminated;

(*c*) animals died of a disease identical with that responsible for the mortality in the owner's cattle on vlei pasturage where no known poisonous plant was present.

In order to exclude, as possible causes of the disease, diverse plants belonging to genera with species supposed to be poisonous to cattle in other parts of the world, these were offered in large amounts to susceptible animals at the Laboratory. The net result was that a species of *Sium* (*S. thunbergii*), gathered from the farm where the experiments were undertaken, proved to be toxic on one occasion. Unfortunately, the animal died during the night and therefore no symptoms were recorded. The post-mortem appearances, however, very closely resembled those described later on. " Sium from the Aapies River " fed to cattle at the Laboratory was harmless.

Other plants reputed to be toxic elsewhere—for example, *Ranunculus* and *Equisetum*—were fed in large quantities to animals without bringing about the slightest traceable disturbance.

These tethering experiments were commenced on 25th October, 1925, the animals being actually confined to the sites given approximately in the sketch-map appearing in the second part of this report. It will be seen that the sites were so selected that the following conditions obtained:—

(1) In sites A, the animal (No. 639) was grazing on vlei vegetation in which both *Sium* and *Equisetum* were present. The animal also had access to water from the washings of the lead-mine.

(2) In sites B, the animal (No. 633) was grazed on vlei vegetation on which there was a little *Sium*, but a very large amount of *Equisetum*. It also had access to water as in (1).

(3) In sites C, the animal (No. 640) grazed on vlei vegetation where both *Sium* and *Equisetum* were absent and at a point above the entrance of water from the mine washings into the stream.

(4) In sites D, the animal (No. 629) was grazed on relatively speaking good grass which contained no vlei vegetation. Moreover, this animal received water from natural fountains on the south ridge.

(5) Sites E were so selected that the animal (No. 632) was confined to vlei grazing free from both *Equisetum* and *Sium*, but the water was the same to which animals in (1) and (2) above had access.

(6) Sites F were so situated that there was a complete absence of vlei vegetation (animal No. 651).

The grazing, however, was relatively speaking poor, and the animal was forced to drink water coming from the lead-mine.

The results of these experiments may be briefly summarized as follows:—

(*a*) The animal on sites A became ill and on 2nd November showed some of the characteristic symptoms to be described later on. This animal, however, refused to take any further vlei vegetation on its first spot, and was consequently moved to another spot where the conditions were more or less identical. The cow improved for a time, but later on became so weak that it was necessary to transport her in an ambulance to Onderstepoort, where she recovered completely.

(*b*) The animals on sites B, D, and F did not show any symptoms beyond a slight diarrhoea.

(*c*) The animal on sites C was extremely excitable and got bogged on several occasions. It is uncertain whether the bogging could be connected with vlei poisoning. This point will be discussed later on.

(*d*) The animal (No. 632) tethered on sites E developed typical symptoms of the disease under discussion within 14 days of the commencement of the experiments and died two days later. On this site, within the tethering range, no single plant of a known toxic nature could be found, e.g. *Sium*, *Equisetum*, and *Ranunculus* were entirely absent.

The above experiments were repeated during the spring and summer of 1926, with results very similar to those obtained in 1925. During the experiments the animal on sites A became ill and typical symptoms were recorded. These tethering experiments were therefore discontinued after a few weeks.

In addition to tethering experiments, feeding tests were also carried out as indicated in Part II. These results may be summarized as follows:—

(1) *Sium*, fed to Cow No. 707, and *Ranunculus*, fed to Cow No. 717, in amounts of two pounds daily did not produce any symptoms.

(2) Vlei vegetation, cut in sites C and E, where *Sium*, *Equisetum*, and *Ranunculus* were absent, and fed *ad libitum*, produced typical symptoms of the disease, the animal dying in one instance and being destroyed *in extremis* in the other. The post-mortem appearances were typical in each case.

(3) On account of these results, five plants which were very prevalent in sites C and E were finally selected for feeding tests and animals fed daily according to the following plan:—

Feeding Tests commenced 12.11.26.

(1) Cow No. 743, fed on 2 lb. *Scirpus corymbosus* + 10 lb. Onderstepoort hay.
(2) Cow No. 605, fed on 2 lb. *Carex cernua* + No. (1) + 8 lb. Onderstepoort hay.
(3) Cow No. 719, fed on 2 lb. *Pycreus umbrosus* + Nos. (1) and (2) + 6 lb. Onderstepoort hay.
(4) Cow No. 633, fed on 2 lb. *Eleocharis limosa* + Nos. (1), (2), and (3) + 4 lb. Onderstepoort hay.
(5) Cow No. 651, fed on 2 lb. *Haemarthria compressa* + Nos. (1), (2), (3), and (4) + 2 lb. Onderstepoort hay.

These feeding tests ended 23.1.27.

The first three cows refused to take their ration after five days. In no instance, however, did nervous symptoms appear. After a week of starvation, the animals again began to feed, and for several weeks nothing but mild laxation could be observed. This laxative effect was probably brought about by the coarse nature of the vegetation causing intestinal irritation.

On 2nd January, 1927, animal No. 605, fed on *Carex cernua* and *Scirpus corymbosus*, showed violent symptoms, during which the right horn was broken. These symptoms, excepting that they were exceptionally violent, were typical of the disease under discussion. On the following day the animal showed peculiar champing movements and grinding of the teeth, together with smacking movements of the mouth. These symptoms disappeared in a day or two.

Animal No. 719, fed on *Pycreus*, *Carex*, and *Scirpus*, also showed fairly violent symptoms during the night of 4th January, 1927. Later on the symptoms subsided somewhat and became typical of the disease as observed under natural conditions. This animal died during the night of 6th January.

These feeding tests were rearranged on 24th January, 1927, two animals (Nos. 4717 and 743) being fed on *Pycreus*, No. 719 on *Scirpus*, a fourth (No. 605) on *Carex*, and a fifth (No. 651) on *Haemarthria* and *Eleocharis*. In each case the animal received its respective feed *ad libitum*.

On account of the lateness of the season and the prevalent drought, these experiments had to be discontinued.

SYMPTOMS.

The symptoms shown by cattle may be briefly summarized as follows:—

(*a*) In acute cases there is inappetence followed by marked nervous disturbance, varying from mania to extreme dullness and even coma. Death, as a rule, follows in a few hours, but recoveries within a few days have been recorded.

(*b*) In average cases the symptoms are approximately as follows:—

The animal becomes hyperaesthetic after a short period of dullness and therefore the herdboys very frequently report viciousness.

Later the animal shows a partiality for shade and grazes with reluctance. These symptoms, however, are due to dilatation of the pupils and a certain amount of paresis of the tongue and cheeks. The symptoms noted above progressively increase, so that in the end the animal is unable to prehend, masticate, and in bad cases even unable to drink. Frothy saliva dribbles constantly from the mouth. During this stage the pupils are widely dilated, and the animal in consequence always makes for dark places. In its progress it may walk straight up against any object in its way, and may stumble over it. Often the animal may even stand pushing against such objects for hours on end apparently because it does not understand how to get away. There are now, as a rule, irregular muscular tremblings, such as flicking of the eyelids, etc. Reflexes are also usually markedly exaggerated.

These symptoms, provided that the animal is fed artificially, may last for a period varying from several days to several weeks. Drenching the animal with concentrates has so far not been found beneficial beyond that it prevents rapid loss of condition and strength.

(*c*) In some cases the train of symptoms is as follows:—

There is a complete inappetence accompanied by extreme dullness. The animal tends to lean against objects or hang in its collar and when forced to move the gait is uncertain. Soon nervous symptoms develop, which vary from convulsive fits to rhythmic contractions of muscular groups, so that the animal appears to be suffering from hiccoughs. At the same time a paresis of the lips, cheeks and lower part of the tongue develops, so that the animal cannot drink or eat in a normal manner. Driven by great thirst, the animal soon learns to drink by dipping the whole lower part of the face into the water. Vision is also impaired, owing to dilated pupils, and after a few days the dullness is succeeded by a hyperaesthesia. Such animals may recover slowly or may die from starvation.

In cases showing symptoms as in (*b*) and (*c*) there is a constant champing of the jaws and frothy saliva dribbles continuously from the mouth. In some cases the tongue is slightly protruded, and in all cases the muzzle becomes soiled during futile efforts at prehension.

(*d*) In the mildest cases mere inappetence with loss of milk and indefinite nervous symptoms have been recorded.

In all cases where the animal did not die, convalescence extended over a period of weeks.

POST-MORTEM APPEARANCES.

The post-mortem appearances are exceedingly variable, and only one constant feature could be found, viz., a very marked and rapidly appearing rigor mortis. This phenomenon was observed in every case, and, as a rule, rigor mortis was present over the entire body within an hour after death. A second change, while not constant, was noted in a large number of cases, i.e. hyperaemia of the meninges and brain. In some cases the hyperaemia was slight, but in other cases very marked.

Apart from the above changes, nothing abnormal may be found at post-mortem. In other cases, again, marked changes may be noted, viz., rigor mortis very marked throughout the body, hyperaemia of the brain and meninges, venous engorgement and slight oedema of the lungs, dilatation of the right ventricle and contraction of the left, both cavities containing uniform red clots, liver considerably enlarged, showing venous engorgement and marked fatty degeneration (and pigmentation), gastro-enteric catarrh, cloudy swelling of the cortex of the kidneys.

In many cases, especially those which are violent prior to death, various traumatic lesions, generally over the prominent parts, may be noted. Finally, it may be added that the carcass is in a state of emaciation when the disease has run a long course.

The post-mortem appearances in the case of ox No. 1179, which died on 27th October, 1925, after receiving 18½ lb. of green *Sium* over a period of three days, were as follows:—

> Hyperaemia of the brain, venous stasis and oedema of the lungs, ecchymoses under the epicardium and right endocardium, and in the mucous membranes of the trachea and bronchi, venous stasis and slight fatty degeneration of the liver and kidneys, hyperaemia of the gastric mucous membranes, tumor splenis.

DISCUSSION.

The data recorded here leave no room for doubt that certain of our South African vleis are poisonous to cattle (and possibly to other stock). We have so far definitely proved that one vlei is certainly very poisonous to cattle under certain conditions. The probability, however, is that vleis are poisonous to a greater or lesser degree over wide areas. There is another vlei some 40 miles away from the one where the recorded experiments were carried out; here a disease with nervous symptoms makes its appearance every year during the spring and dry summer months. At first it was thought that heartwater was responsible for the symptoms and deaths, but transmission experiments by blood inoculations gave negative results in every case.

Apart from the case mentioned above, farmers from practically all over the country complain about a disease popularly called dronk galsiekte (drunk gallsickness, especially during the spring and dry summer months. We usually took it for granted that dronk galsiekte was nothing but heartwater. In quite a large number of cases blood inoculation into susceptible animals failed to confirm this.

With the information gained during the last two seasons it seems probable that many cases, diagnosed as heartwater in the past, were actually cases of vlei poisoning, similar to, if not identical with, that recorded here. It may be mentioned that dronk galsiekte is also reported by farmers from localities where heartwater is unknown.

In some respects the symptoms of the vlei poisoning under discussion also somewhat resemble those of lamsiekte, especially where paresis of the cheeks and tongue are very marked. In lamsiekte, however, other nervous symptons are never noted, and their absence serves to differentiate between it and vlei poisoning.

The toxicity of the vlei under discussion varies very considerably from month to month. The fact that cattle free to roam where they like contract the disease only during the spring and early summer months, is perhaps due to better and more palatable grazing after the early summer rains; such cattle then undoubtedly eat less of the coarse vlei vegetation. To put this differently, during the spring and early summer months the green vlei attracts cattle, and they then eat a very much greater bulk of the plants growing there than later on when good grazing is available. But with the tethering and feeding experiments the cattle had no liberty to roam, and yet the toxicity was different from time to time, and even from place to place, e.g. in 1925 the animals on A sites became ill first, but recovered, whereas the animal on E sites was the first to die after showing typical symptoms. In 1926 the A sites again were responsible for the first symptoms, but plants from the C sites caused the first death, those from sites E only producing typical symptoms considerably later. Again, in the feeding tests, nothing abnormal happened during the period 12.11.26 to 1.1.27, and then suddenly two animals became ill, one recovering and the other dying.

There is nothing startling in this, for several of our South African poisonous plants give similar results, e.g. Gifblaar (*Dichapetalum cymosum*), *Tribulus terrestris*, *Vangueria*, and *Sorghums*. In the case of gifblaar, the plant contains its dangerous toxic principle in large amounts only during its two periods of active growth at and about the equinoxes; *Tribulus* again is only dangerous during a restricted time of the year, and *sorghum* sprouts are most dangerous during dry spells. The circumstances that determine the varying toxicity of *Vangueria*, etc., are not fully understood.

In the case of vlei poisoning, one or more of the causes enumerated above may determine the toxicity of the vlei plants. When this varying toxicity was realized, it was too late to make observations or carry out experiments in connection therewith. This, however, will form the subject of a future study. One thing seems certain, however: that the physiological processes of the plants vary under varying conditions, and that substances of a toxic nature are only produced in large amounts under certain conditions, whatever these may be.

It cannot be said that the toxic plants have been definitely identified. *Sium*, however, is definitely poisonous under certain conditions, namely, those prevailing during 1925. Under the conditions prevailing during 1926 it proved to be harmless to cattle. This, however, is not an uncommon experience with South African poisonous plants, e.g. *Geigeria passerinoides* (vermeerbossie), *Vangueria* (gousiekte bossie), and *Tribulus terrestris* (dubbeltjie).

The phenomena here recorded are common to very many South African poisonous plants. A long incubation period cannot be assumed in the case of some poisonous plants, e.g. *Sium* sp. On the other hand, again, most of the experimental animals that got ill only showed typical symptoms after a week. This can be interpreted in many ways, e.g.:—

(1) That there is a definite incubative period. The experiments, however, do not support this view.

(2) That there is a cumulative effect of the poison. The experiments to date do not clarify this point. Indeed, the matter can only be solved after the poisonous plants have been identified beyond any question of doubt.

(3) That there is at least one plant other than *Sium* which under certain climatic (or other) conditions may be poisonous to cattle. From the feeding tests carried out so far it is unfortunately impossible to say which of the five plants mentioned in the text is certainly responsible for the symptoms and post-mortem appearances recorded. In view of the fact, however, that animals receiving *Scirpus* plus a small supplementary ration become ill, this plant comes under very strong suspicion. In the early part of this article the excitability of the animal tethered on site C was mentioned. In this locality there was an excess of *Scirpus*. It is possible that the nervousness of the cow was due to vlei vegetation rather than to natural tendencies. If this supposition is correct, then *Scirpus* would receive a very large amount of support for its varying toxicity. The other two plants, namely, *Pycreus umbrosus* and *Carex cernua*, cannot be excluded as possible toxic agents. The feeding experiments undertaken at the present time may serve to elucidate this point later on.

The fact that vleis are poisonous in certain localities and under certain circumstances cannot be disputed. The poisonous plants have so far not been identified except in the case of Sium. There seems to be every reason to suspect that the normal physiology of a plant may be so disturbed as to lead to the production of products primarily poisonous or poisonous upon digestion. The well-known cases of *Sorghum* poisoning illustrate the possibility of the above suggestion.

The case, however, has by no means been proved and a great deal of investigation, especially with reference to varying conditions, has still to be carried out. This can be done best of all by the field officers.

In conclusion, it may be said that this nervous disease can often be mistaken for heartwater, a disease which appears in low-lying parts. In heartwater, however, there is always a marked hyperthermia as soon as visible symptoms appear. In vlei poisoning, on the contrary, no hyperthermia of any marked degree makes its appearance until muscular contractions are severe.

SUMMARY.

(*a*) A cattle disease occurring during the spring and summer months on certain vlei pasturage is described.

(*b*) The symptoms are mostly nervous and may be confused with such well-known diseases as lamsiekte and heartwater.

(*c*) Post-mortem appearances are indefinite, but rigor mortis always follows very rapidly after death and may be regarded as characteristic of the poisoning.

(*d*) The cause of the disease is vlei vegetation, but the plant, or plants, responsible have not been definitely established.

(*e*) On one occasion a species of *Sium* caused the death of an ox which was fed with this plant *ad libitum*.

REFERENCE.

W. H. ANDREWS, D.Sc., etc. "The so-called 'Staggers' or Pushing Disease of Cattle in Natal," published in 1924 in *9th and 10th Reports of the Director of Veterinary Education and Research.*

Photo of the extended vlei vegetation.

15th Annual Report of the Director of Veterinary Services, Union of South Africa. October, 1929.

Vlei Poisoning.

PART II.—ECOLOGY.

By A. O. D. MOGG, M.A. (Cantab.), Research Officer, Onderstepoort.

THE farm on which the poisoning occurred is situated about 18 miles E.N.E. of Pretoria, just at that point where the Magaliesberg Range (which runs almost due east and west in the Pretoria district) is diverted from its course sharply southwards. There is consequently some faulting and much confusion in the underlying rocks.

This record is concerned mainly with the low-lying paddock at the west extremity of the farm. In order to enable the reader to picture the paddock in its place on the veld and to form an accurate concept of the important factors which influence its floral composition (e.g. soil, drainage, etc.), a brief account of the relationships of the farm to the surrounding physical features will now be given.

The farm is in general shape an oblong whose long sides are represented by two low parallel ranges of hills forming the northern and southern boundaries of the farm, whilst the main area enclosed consists of the valley between, perhaps a mile wide.

These ridgelike hills are of stratified hard quartzitic sandstone overlying softer shales, and comprise part of the Magaliesberg group of the Pretoria Series of the Transvaal System. The strata dip north at a fairly steep angle (about 35°), but the strike slopes are far more gradual (perhaps under 20°) and incline inwards towards a small intermittent stream, which lies in that third of the valley nearer the south ridge.

The east end of the valley is at a much higher level than the western boundary. Therefore, drainage is rapid from east to west, but more gradual inwards on either side from the northern and southern ridges.

The soils on the northern banks of the stream, derived ultimately from the breakdown products of the shales and diabases of the strike slopes, are mainly clayey and very shallow, whilst those on the southern banks and slopes are mainly sandy and deep (of white quartzite origin).

Now our paddock lies in that portion of the farm where the valley broadens and flattens out.

Hence the eastern portion of the paddock displays the streamlet with steep shelving banks cutting a narrow deep meandering channel through black clayey alluvial soil, exhibiting the force of the torrent during the spate periods of the summer rainy season; whilst in the western portion of the paddock the stream bed rapidly broadens out fan-wise into a marshy inundatable flat area over 100 yards wide. Here the mixed transported soils are deposited to great depths and richness, and carry correspondingly luxuriant hydrophytic vegetation.

The delimiting western boundary of the farm, and thus of our paddock, consists merely in a wire-fence running from north to south across the valley. Midway between the valley stream and the northern hill is a public road, which with the stream thus divides the valley into three almost equal parallel strips.

The western portion of the northernmost strip (that directly contiguous with our paddock and separated also only by wire fences) is of interest to us only in so far that the farmer winters his cattle in this purely hilly-ridge paddock, and the grazing seems harmless. It is only when he moves the cattle from this camp into our vlei-containing adjoining paddock, that " vlei poisoning " develops.

Our paddock is a rectangular fenced camp which extends across the strip between the road and the stream to a fence about 200 yards beyond the stream on the southern side, and about half a mile up the valley from the western extremity in an easterly direction. (See plan, page 830.)

The north-eastern portion of this valley paddock was cut off by a fence running south-west obliquely from a point on the road below the residence, almost to the stream, and thence due east almost to its eastern boundary fence.

Within this portion is a small lead-mine. This mine has been worked at irregular intervals for about 30 years. Galena is found within a few feet of the surface. The water from the old mine workings flows constantly into the valley stream, and when ore is crushed and washed the slimes drain into the stream at a point very near the eastern extremity fence. (See plan, page 830.)

SOILS.

Those on the northern side are derived from the weathering of the underlying shales and diabase, together with a superficial admixture of sand ultimately derived from the breaking down of the quartzite screes. Hence the consistence is clayey; the more so, the nearer the stream. The soil, moreover, is very thin (so much so that the underlying rock is often exposed) and bakes hard. It carries typical southern slope, high-veld ridge vegetation—Andropogonous grassland with occasional low shrubs—but rainwater does not readily penetrate this baked surface, and is usually largely lost. Nearer the stream a large group of thorn trees occurs (Acacia karroo), quite dense in parts, and as is frequently the case, they indicate subterranean water. This local consociation is strongly marked and carries its own associated species.

The soils on the southern side of the paddock are, however, very different. These are derived from the steep quartzite, scree-covered slopes which occur in their close proximity. Hence they are predominantly sandy, and where untouched, carry a flora typical of many miles of the Magaliesberg northern talus slopes. *Stoebe cinerea* is a dominant suffructicose herb in this consociation and the grass *Perotis latifolia* occurs as a typical associated species in this region.

Hence on either side of the paddock stream dark, rich, fine alluvial soil has been deposited, often to great depth (just below the mine dumps, the stream cuts channels quite 10 ft. deep into it). This is mainly clayey in consistency.

As might be inferred from the topography outlined, the more westerly portion of the paddock would tend to be more heavily silted up, which is the case. Also the angle of slope from east to west in this portion of the valley is greatly lessened, and the stream, having already cut deeper channels in the easterly portion of the paddock, can therefore flow but sluggishly in the western portion of the paddock, resulting in greater deposits of suspended matter. Concurrent with the lessening of the slope from east to west, the slopes from either bank are here also relatively more gradual. The consequence is that the stream, usually a couple of yards wide, broadens out into a marsh in parts nearly 80 yards wide throughout the western third of the camp.

It is with this stream-side vegetation, and particularly the marshy portions, that we are mainly concerned.

HISTORY.

The history of this farmer's experience with disease in his cattle is given fully by Neser. Suffice it to say that the farmer virtually gave up stock farming and explored the possibilities of agricultural farming. The catchment area of the stream, including several strong springs about 2½ miles from the western fence, was dammed up, and the deep soils of the valley were ploughed up and planted, mainly on the south side, right up to the more sandy patches which were irrigable, at the base of the southern ridges.

Thus a large portion of the camp with which we are concerned was ploughed, and on the southern and eastern sides there still remains arable land under cultivation. This disturbance of the valley vegetation occurred about seven years ago.

As, however, crop production did not entirely satisfy the farmer's tastes and inclinations, he soon fallowed the vlei and gradually acquired a fresh herd of dairy cattle, which were put to graze on this altered valley vegetation. In 1924 these animals began to show symptoms of a new disease, which led to these botanical observations to supplement the work of the veterinarian.

The next step, therefore, will be for us to examine the composition of the vlei vegetation, and some of the more important changes it has undergone in recent years.

ECOLOGY.

By studying the lower swamps owned by neighbours to the west of our paddock, and by making careful notes of the stream-course to beyond the eastern dam, much of the previous composition of that hydrophytic association can be fairly accurately estimated.

There is little doubt that the whole stream-course was densely colonized by reeds (*Phragmites communis*), bulrushes (*Typha australis*), and the mat sedge (*Scirpus corymbosus*), the *Typha* forming the innermost zone and the *Phragmites* the outer. Typical undisturbed associations of this type occur to-day two or three miles down-stream. Wherever the area was muddy and waterlogged, *Typha* predominated, *Scirpus* also being very prevalent. However, wherever the area was drained although wet, the reeds predominated to the exclusion of almost everything else, as they do in the case of running water.

Associated with the reeds, on similar, drained, wet soil, and growing amongst them, rather on their outer side, occurred *Carex cernua*, a cyperaceous plant with very sharp saw-edged leaves. Just without this zone, but commingled with the *Carex*, occurred the zone of *Pycreus umbrosus*, a shiny leaved, more low-growing sedge.

That, so far, would be a normal zonation for a highveld vlei and stream of this type. Had it been left in the probable state it was in prior to 1918, that, in essential features, would be what we would find to-day.

It should here be noted that all these species are deeply rhizomatous and would largely tend to persist even although surface conditions might alter fairly considerably, e.g. become drier or colder.

The next series of zones occupied relatively drier ground. The first was composed chiefly of *Eleocharis limosa*, together with *Haemarthria compressa*, the latter in a fairly broad belt, depending upon the angle of slope. Beyond this occurred *Fimbristylis complanata*, well up the stream banks and merging with almost dramatic suddenness into *Andropogon eucomis* and *Imperata arundinacea*.

In the muddy stream-course, mostly semi-submerged, one found the umbelliferous *Sium thunbergii*. The edges of stream-courses are apparently the euhabitat for this species, whose rhizomes entrench themselves deeply. For if extensive silting-up occurs during spate periods, the *Sium* remains, emerging above it with reduced leaves and rosette habit. Indeed, these persistent remnants occupy a prominent part in these investigations, for although they are of interest as indicating the former courses of the stream (and possibly of a present subterranean flow), yet, being toxic, this species occurs in the zones most closely grazed by cattle, which seek out the luscious *Haemarthria*.

Similarly on the banks, especially in the sand silted-up areas, occurs *Equisetum ramosissimum*. This plant is irregularly distributed and in small quantities; its interest lies in its being so persistently suspected of being toxic. No proof of this whatever exists. However, there appears to be abundant evidence for the toxicity of closely allied species, natives only of Europe and America.*

Of the semi-floating plants, *Scirpus fluitans* is of interest; the rhizomes, rooted in the mud of the water's edge, branch repeatedly and produce enormously long stems, which mostly float on the water and which at short intervals send up erect inflorescence stalks.

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* See "Plants Poisonous to Live Stock," by H. C. Long, Chap. VI, and "Manual of Poisonous Plants" (Pammel), Part II (1918).

Of the submerged plants, a species of *Nitella* is the most prevalent and conspicuous.

The full list of species about the vlei in the paddock will be given in the floristic lists for each experimental animal tethered on selected areas about the stream and marsh. (See lists, pages 827-829.)

I have mentioned the chief zones which most probably existed before the camp was disturbed by ploughing, fallowing, and extensive grazing. But a very different composition was found to be present in October, 1924. Essentially, of course, the zonation remained the same, but the proportion of the constituents was entirely altered, so much so that whole species and their zones, including even the dominants, were found to have almost completely disappeared so that the entire facies of the vlei was altered. Thus only remnants, a specimen here and there widely separated, of the reeds could be found. *Typha* was almost non-existent, and the facies of the central vlei was altered to one of low aspect from 1-2 ft. high, instead of the dense, overshadowing, exclusive, wandlike forms. The result was that *Scirpus corymbosus*, not being overshadowed and crowded out, and, moreover, occurring in the most boggy part of the vlei where stock were least likely to eat it down, was found in very great abundance, i.e. to have increased to abnormal proportions and become the dominant.

Similarly *Carex cernua* was found unusually prevalent and tall, and occupying habitats it never could have retained had the reeds not disappeared from their zone. But the most conspicuous change, which incidentally one has observed in many other altered vleis, particularly burned vleis, was the enormous proportion of *Pycreus umbrosus*. Now this species is capable of living in very dry soil, as well as even in the semi-submerged portions of a vlei, the depth and number of the rhizomes being altered to suit the conditions. So facile is this species in its capacity to range that, given the absence of serious competition on either the water or land side, it can, in a single favourable season, usurp almost the whole area. Hence it is often a valuable index of changed vlei conditions, particularly of seasonal variations.

Normally in undisturbed high veld vleis it is limited on the water side by the *Juncus-Scirpus corymbosus* zone, and on the land side by the *Eleocharis-Haemarthria-Andropogon eucomis* zones. In 1924 I found this species well beyond these zones on either side, and this condition obtained along the greater part of the stream-course.

In the broad vlei portion, where the angle of slope from the water is so gradual that the zonation of species is enormously broadened (see page 830, Transsects. V and VI) one found *Pycreus* from the water's edge to the *Andropogon eucomis* zone, i.e. throughout the whole breadth of the vlei.

In October, 1925, the condition above outlined had increased, particularly with regard to the *Scirpus corymbosus* and the *Pycreus umbrosus*.

The farmer, as in previous seasons, had grazed his stock in the northern hill paddock until about the beginning of August, when they were turned into this vlei paddock. From about the beginning of October typical cases of the disease began to appear amongst the cattle.

I went to make a survey of the vlei on the 19th, the day after the third animal had died (the third that season and all since 1st October). As the result of my report, and observations of his own, Neser decided to institute tethering experiments at once, and invited me to assist him in the selection of the sites for his six experimental bovines. This was completed on 25th October.

When tethered, the animals could move freely in a circle of about 30 feet in diameter.

The only other food material the animals received was water, and then only for those animals not within reach of water. (See Neser's account for complete details.)

The animals were left on the sites until, in Neser's discretion, the area had been closely enough grazed.

The prime considerations which actuated me in the selection of these sites were as follows:—

A. The inclusion of as great an aggregation as possible of any suspected toxic species.

B. The securing of at least a preponderance, within the tether area, of each important marsh constituent.

In the floristic lists I had previously made, no plant occurred about which authentic information was available as to its toxicity. The two plants, *Sium thunbergii* and *Equisetum ramosissimum*, were strong suspects on account of reliable records of toxicity in closely allied species of these genera both in America and Europe. Hence all zones had to be exploited, including, of course, the ridge vegetation as well.

Therefore: (1) Sites A1, A2, and A3 were selected and pegged, so as to include chiefly the stream (semi-submerged) zone, but such portions of it as contained an abundance of both *Sium* and *Equisetum*. (See protocols of floristic lists, pages 827-829.)

(2) Sites B1, B2, B3 were selected on the silted banks and inundatable areas containing an abundance of *Equisetum*. (See page 830, Transsect. IV.)

(3) Sites C1, C2, C3, stream and stream-side vegetation (mainly *Pycreus umbrosus*), above where the mine-washings water entered the stream (in case lead should be involved) and excluding *Sium* and *Equisetum*. (See page 830, Transsect. I.)

(4) Sites E1, E2, E3, etc., were selected in the broad vlei zone, well below where all the mine-washings came in (hence all the semi-submerged plants were constantly immersed in it), but excluding *Sium* and *Equisetum*. These tethering sites consisted of almost equal admixtures of *Pycreus umbrosus* and *Carex cernua*. (See page 830, Transsect. V.)

(5) Sites D1, D2, D3, etc., and F1, F2, etc., to test the south (sandy soil) and north (clayey soil) ridge vegetation respectively.

Floristic lists were made of the species and their visual prevalence in the tether circle, and also observations were recorded of the species grazed, avoided, etc. This information for all animals and for all species is collated on one sheet. (See pages 827-829.) By this means reference to the list of the tethered area of any animal exhibiting symptoms of the disease, would at once indicate and emphasize a comparatively few plants in the whole vlei flora.

After the experiments had been in progress a short while, the bovine (No. 639) on Sites A sickened. Later, the bovine (No. 632) on Sites E died. Also a feeding test carried out at Onderstepoort Laboratory, where a bovine was fed almost exclusively on fresh *Sium* brought from this vlei, gave positive results since the animal died.

Now if we examine the " incidence " of species in E's list (bovine No. 632) (see photo), it will be seen that *Pycreus umbrosus* is the most abundant species, and remains the most prevalent common factor if taken in conjunction with bovine A's (No. 639) list; *Haemarthria* comes next, then *Scirpus cernua*—all *readily eaten* that season, with the exception of the latter, which was usually only accidentally eaten.

It will be noticed that all these species were in the young, spring state. It is true that sporadic flowering occurs with *Pycreus*, but none of the principal species cited flower until much later. *Scirpus corymbosus* flowers best in November-December, but all the others flower later than that, January-February being their optimum flowering periods.

No further marked symptoms having occurred by December, the experiment was discontinued for that season.

The farmer was also advised to keep his stock away from that vlei, and the area was in consequence fallowed until July, 1926.

This had a considerable effect on the vegetation: on the water side, *Scirpus corymbosus* increased and became tall and dominating. The entire facies of the vlei was governed by this type.

Concurrently and coincidentally it was necessary for *Carex cernua* to grow vigorously and tall to combat and compete with this invasion. Such a response indeed was made, fortunately favoured by late seasonal rains.

The effect on *Pycreus* was considerable. All its stores of food were devoted to producing long laminae to avoid being shaded out, and hence radial spread was not only checked but largely prevented. In fact, on the water side, the invasion of *Carex* was too strong for it, and it was almost ousted. For example, the site of E1 was changed from a proportional representation of about *Carex* 35 : *Pycreus* 65 to *Carex* 60 : *Pycreus* 40 ratios.

Similarly on the land side, *Eleocharis limosa* and, on its outer zone, *Fimbristylis complanata*, both increased to an extraordinary extent, limiting the former spread of *Pycreus* and in the portions of the vlei of low slope angle (see page 830) Transsect. I and VI) much of the usurped area was even reclaimed from *Pycreus*.

Andropogon eucomis also increased in abundance. It might be noted that these observations recorded for *Pycreus* are true also for the low-growing, mat-forming *Haemarthria compressa,* which inhabits the zone adjoining *Pycreus* on its land side.

In the *Haemarthria* zone occurs *Ranunculus pinnatus,* Poir (=*R. pubescens,* Thunb.), although in relatively small quantity, and it is little grazed by cattle.

In mid-September, 1926, Neser decided to repeat the tethering experiment on the same sites; but in addition he also decided to have two animals stabled, these animals to be fed entirely on hay cut from specified portions of the vlei: one to be fed on green vlei hay cut in the broad zone of *Carex-Pycreus* just below bovine E's site (see page 830, Transsect. V), the other to be fed on green vlei hay cut above bovine C's site.

Shortly after this seven more bovines were drafted into the experiment. These were all tied up separately under the shade of trees, on cleared sandy soil, and their food was placed into low wooden boxes, borehole water being their only other ration.

The food which was presented to them in the boxes consisted of specific plants cut from the vlei. The species selected were chosen by me from the common factor lists for sites A and E of the 1925-26 season (see pages 827-829), and the order agreed upon was (1) *Scirpus,* (2) *Carex,* (3) *Pycreus,* (4) *Eleocharis,* (5) *Haemarthria.*

Neser arranged the manner of presentation as follows:—

(1) Bovine No. 743 to be fed 2 lb. *Scirpus corymbosus* + 10 lb. dry Onderstepoort hay.
(2) Bovine No. 605 to be fed 2 lb. *Carex cernua* + No. 1 + 8 lb. Onderstepoort hay.
(3) Bovine No. 719 to be fed 2 lb. *Pycreus umbrosus* + Nos. 1-2 + 6 lb. Onderstepoort hay.
(4) Bovine No. 633 to be fed 2 lb. *Eleocharis limosa* + 1, 2, 3 + 4 lb. Onderstepoort hay.
(5) Bovine No. 651 to be fed 2 lb. *Haemarthria compressa* + 1, 2, 3, 4 + 2 lb. Onderstepoort hay.

Two bovines were fed with an *ad libitum* ration of *Sium thunbergii* (bovine No. 707) and *Ranunculus pinnatus* (bovine No. 717).

The manner of collecting was as follows: The food material was obtained by two natives with sickles and sacks; the portions of the species that would naturally be grazed by bovines were sickle-cut or hand-plucked from the growing plants in specially selected streamside strips. The natives were trained to choose the correct plants, placing these culls into damp bags, and this work, together with the accurate subdivision of the sacks' contents into the scheduled food lots, was personally supervised by the farmer himself. Further, to assist in the elimination of errors, a bunch of each feeding test species was fastened in a convenient conspicuous place immediately above the correct numbered bovine.

In October, the bovine on site A died with alleged typical symptoms. This might have called for a different arrangement of the order of the feeding-schedule for the above five bovines. However, nothing was done, it being anticipated that other cases might develop.

Soon the feeding tests with *Ranunculus* and *Sium* were discontinued. On 11th November the bovine No. 3174 suddenly developed symptoms, and died on the 12th November with what the farmer considered to be the typical symptoms of the vlei poisoning he had experienced in his own previous cattle losses.

On visiting the farm the next morning, with Lawrence, of the Division of Veterinary Services, I estimated the rumenal contents of this dead animal to consist of about 95 per cent. of *Pycreus umbrosus*, one or two fragments of *Scirpus*, and one or two pieces each of *Sium* and *Equisetum*.

The uneaten food in the trough, quite two days' rations, was of about the same composition, i.e.—

Pycreus	95 per cent.
Scirpus	2 per cent.
Equisetum	2 per cent.
Sium	0.5 per cent.

There were no other constituents.

Now it must be noted that the feeding material had been previously collected from the hydrophytic zones *above* the sites C of bovine No. 640. But the supply had given out, and on the 3rd November, Neser had ordered the food materials to be cut *below* site C (bovine No. 640) (see page 830, Transsect. II) in the streambed where the mine-washings silt of last season comprised at least the first six inches of soil. Here the vegetation was rank, being composed of deeply rhizomatous and semi-submerged hydrophytes. *Scirpus corymbosus* was dominant and grew very tall; but *Pycreus umbrosus*, with very long coarse laminae, was the most prevalent species. This zone is the euhabitat for *Sium*, and in the more raised silted-up areas *Equisetum* was very abundant, particularly in that very elevated portion where the mine-washings fell in a miniature waterfall into the pool.

Now the natives, in cutting the rations, had rejected the very coarse *Scirpus* stems (hence the small proportions found in the feeding trough and rumenal contents), leaving them on the cut area. This also applied to the *Sium*. In the actual strips cut very little *Equisetum* grew (although it was relatively very abundant in the near vicinity).

Had one not closely examined the rumenal contents, one could not have excluded the possibility of the animal having selected out and eaten more *Sium* than would be suggested by the proportion in the troughs. But only two leaves in the whole rumen could be discovered.

Hence three points stand out with this death: (1) *Pycreus umbrosus*, at least for the last 3-4 days, composed over 90 per cent. of its rations. (2) The animal was apparently healthy (and somewhat wild), and at least as healthy as bovine No. 640 on sites C last season, whilst it was being fed on hydrophytic vlei vegetation cut on *sites C* (bovine No. 640) and *above*, therefore, where the mine-washings entered the stream. (3) It only died some 8-9 days *after* its rations were changed to hydrophyte vegetation growing amongst deep mine-washings silt. The alteration in site of collection *may* not have anything to do with the inherent toxic principles of a particular plant; but this case does seem to indicate that the effect of the soil factor must not be left out of account.

As the result of this death, the schedule for feeding bovines was altered as follows:—

(1) Bovines Nos. 4717 and 743 fed with '*Pycreus*' *ad libitum* + Onderstepoort hay as required.
(2) Bovine No. 719 fed with '*Scirpus*' *ad libitum* + Onderstepoort hay as required.
(3) Bovine No. 605 fed with '*Carex*' *ad libitum* + Onderstepoort hay as required.
(4) Bovine No. 651 fed with '*Haemarthria*' and '*Eleocharis*' + Onderstepoort hay as required.

This is exactly in accordance with the common factor lists for sites A and E, and also takes into account the proportions just given for the mine-washings silted-up pool below sites C.

Of the species 1-5, none of these plants have a previous toxic record.

Sium is toxic, but possibly only on some vlei soils, and then only perhaps in the spring and early summer. Had the vlei not been so altered and heavily grazed down, it is very improbable that *Sium* would have been grazed naturally at all. It is almost inaccessible to stock in its euhabitat in undisturbed vleis (marshes). The paddock treatment undoubtedly emphasized such plants as *Sium*.

Both *Ranunculus* and *Equisetum* are genera with definitely toxic species elsewhere than in South Africa. So far, *Equisetum* has proved negative in feeding tests, both on this area to bovines, and at Onderstepoort to horses, cattle, and sheep. Similarly *Sium* has not *always* proved toxic, nor has *Ranunculus* at all to bovines.

But as regards this latter species, it is worthy of note that in several experiments carried out by Le Roux, of the Division of Veterinary Services, at Ermelo on *sheep* in November, 1926, death was produced by forcibly feeding (i.e. drenching) the chopped and crushed flowering and fruiting plant. Shortly after this, as fully recorded by Neser, the tethering experiments were discontinued, and only the tests relating to bovines Nos. 4717, 743, 719, 605, and 651 were proceeded with.

These were continued with until the end of January, 1927, when, no further " cases " of any kind occurring, the experiments for this season (1926-27) were concluded.

SUMMARY.

(*a*) " Vlei poisoning " is the term applied to some hitherto unexplained deaths amongst the dairy herd of a farmer in the high-veld in the Eastern district of Pretoria. The animals were grazing in a small paddock containing a permanent stream whose meandering course often included much inundatable, marshy areas.

(*b*) Geologically the portion of the farm concerned is situated in a valley formed between a rather confused portion of the Magaliesberg section of the Pretoria series of the Transvaal System. Hence the higher ground to the North and South sides of the central stream consists of clay and sandy soils respectively, and as such carry very different edaphic, xerophytic plant communities.

The soil immediately about the stream and vlei portions is deep, dark, alluvial, clayey silt soil, derived not only from the relatively slight drainage inwards from the north and south slopes of the valley, but chiefly from the main valley drainage from east to west.

(*c*) Fenced off from the North Eastern corner of the paddock is a small lead-mine which has been sporadically worked for 30 years. The washings from the crushed ores run down open furrows into the stream at points near the eastern boundary fence. Hence some of the products of the mine workings are accessible to cattle in the paddock, and the vlei vegetation grows amongst the clay soil much admixed with this mine silt so that the hydrophytes are steeped in water partly derived from the mine.

(*d*) The cattle deaths have been occurring annually, (1) in the spring and early summer, and (2) since the vlei was ploughed, cut, and closely grazed.

(*e*) By examining the stream-course for a long distance both Eastwards and Westwards beyond the confines of the paddock, abundant evidence was found of the probable former composition of the vlei—not seen by the writer in its undisturbed state. The association was undoubtedly a Phragmitetum-Typhetum stream and swamp—tall, wandlike forms with dense aggregations in the more marshy portions. But by ploughing, cutting, and grazing down the valley vegetation, the whole facies of the vlei portions have been changed. The association dominants have almost disappeared and have been completely replaced by a Scirpetum-Pycretum association, relatively low-growing, non-shading forms.

(f) Undoubtedly owing to the veld treatment, plants have been made accessible to cattle, and have been eaten by them, which would not otherwise have been the case. One such associated plant, *Sium thunbergii*, has been definitely proved to be toxic, at least in the spring state on this soil. Likewise two other plants, *Equisetum ramosissimum* and *Ranunculus pinnatus* (the latter found to be toxic to sheep when drenched), had both been brought into unusual prominence, and were eaten by cattle. Feeding tests to cattle with these species in their spring-summer states, carried out by Neser, have so far proved negative.

(*g*) A survey of the farm paddock having revealed no specific plant with a previous authentic toxic record, Neser decided to initiate tethering experiments. Sites representing different zones of the paddock vegetation were selected by me, and the experiments commenced in October, 1925. Floristic lists, with visual colonization factor notes for each species, were made by me for every animal and site.

Two animals in these experiments became affected: (1) The bovine (No. 639) on A1 sickened on the site where *Sium*, *Equisetum*, and *Ranunculus* were exposed (albeit *Pycreus umbrosus* and *Scirpus corymbosus* were the most abundant species present and eaten). This case led to the *Sium* test recorded above. It had long been suspected to be toxic, but never proved.

(2) The bovine (No. 632) on E1 sickened and died. Its tether site conspicuously had neither *Sium*, *Equisetum*, nor *Ranunculus*. The main grazing (on alluvial inundatable sandy silt soil at the

commencement of the broad marshy zone) consisted of 1, *Pycreus umbrosus*; 2, *Carex cernua*; and 3, *Scirpus corymbosus*.

(*h*) The experiments repeated in Spring, 1926, showed two cases, (1) one tethered on site A1, and (2) one stabled bovine fed from green hay cut from the rank stream-bed growth. The ration composition was examined (A) in the field, (B) in the uneaten remains in the trough, and (C) in the actual rumenal contents of the dead animal, showed that *Pycreus umbrosus* was present over 90 per cent., and only minute proportions of *Scirpus*, *Sium*, and *Equisetum* were present.

(*i*) Feeding tests carried out by Neser on cattle away from the vlei and drinking borehole water, but being fed by specific plants cut from the vlei fresh daily, proved negative.

(*j*) It is noteworthy that the animals which either died or only sickened were (1) drinking mine-contaminated water and (2) eating vlei and streamside vegetation growing in or near and also *below* where the mine-washings entered the stream. Whilst animals on the ridges and animals in the vlei *above* where the mine-washings entered the stream, throughout remained unaffected.

Also, not all portions of the vlei, even below the mine, are equally toxic (e.g. site B in both seasons, and site E this season).

CONCLUSIONS.

(1) Vlei poisoning seems to occur only during the spring state of the hydrophytic portions of certain parts of one particular high-veld vlei, cattle only being affected.

(2) One essentially vlei constituent, *Sium thunbergii*, which was enormously emphasized owing to the treatment which modified the vlei, was proved toxic in the spring on that soil.

(3) That *Sium* was not the only cause of death, but that (*a*) *Pycreus umbrosus* chiefly, then (*b*) *Scirpus corymbosus* and (*c*) *Carex cernua*, were principal constituents of the food of all affected animals.

(4) That all the feeding-test plants were growing in that portion of the vlei below where the mine water and ore washings would enter and contaminate the stream. No experiments have been devised or tried which would completely eliminate the possibility of mine products or soil changes from influencing the vegetation and so eventually being concerned with the cause of the vlei poisoning.

VLEI (VALLEY) POISONING INVESTIGATIONS: COMPARATIVE FLORISTIC LISTS.

	25th October, 1925.	Bovine No. 639.	Bovine No. 633.	Bovine No. 640.	Bovine No. 629.	Bovine No. 632.	Bovine No. 651.	Total Incidence.	Graz-ability.	A. & E.
		A. Vlei.	B. Vlei.	C. Vlei.	D. S. Ridge.	E. Vlei.	F. N. Ridge.			
	SPECIES.	Water Zone (*Sium-Equisetum*).	Silted Zone (*Equisetum* dominant).	Eastern Vlei Zone.	Sandy.	Western Broad Zone.	Clayey.	Frequency of Species.	Ob-served.	Critical Floristic Lists.
Equisetaceae.......	*Equisetum ramosissimum*, Defs.........	C.	A.	—	—	—	—	—	E.	—
Typhaceae.........	*Typha australis*, Schum and Thon......	L.	—	—	—	—	—	—	T.	—
Gramineae........	*Cynodon dactylon*, Pers................	R.	AD.	—	—	—	L.	—	SE.	—
	Digitaria eriantha, Steud., var. *stolonifera*, Stapf........................	—	—	—	A.	—	—	—	RE.	—
	Eragrostis brizoides, Nees..............	—	—	L.	C.	—	—	—	RE.	—
	Eragrostis plana, Nees.................	C.	FC.	C.	C.	L.	—	AD. 5 L.-C.-AD.	E.	L.
	Haemarthria compressa, Poir...........	A.	L.	A.	A.	A.	—	5 L.-A.	RE.	A.
	Imperata arundinacea, Cyril............	C.	VC.	—	—	L.	C.	4 L.-C.	E.	L.
	Leersia hexandra, Swartz..............	L.	—	—	—	C.	—	—	SE.	L.
	Paspalum dilatatum, Poir..............	A.	A.	A.	VC.	—	—	4 A.	RE.	—
	Pennisetum thunbergii, Kunth..........	L.	—	C.	—	L.	—	—	SE.	L.
	Phragmites communis, Trin............	R.	—	—	—	R.	—	—	T.	R.
	Sporobolus fimbriatus, Nees.............	—	—	—	—	—	F.	—	E.	—

VLEI (VALLEY) POISONING INVESTIGATIONS: COMPARATIVE FLORISTIC LISTS—(*continued*).

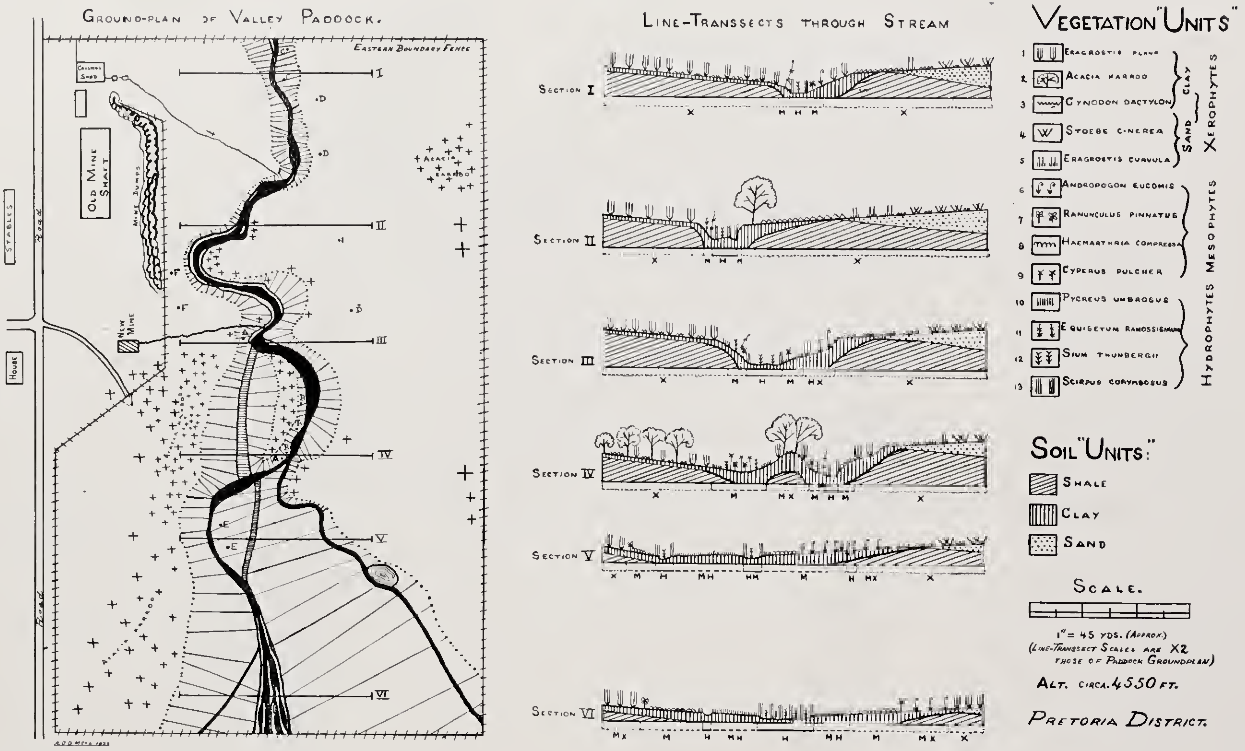
	25th October, 1925.	Bovine No. 639.	Bovine No. 633.	Bovine No. 640.	Bovine No. 629.	Bovine No. 632.	Bovine No. 651.	Total Incidence.	Graz-ability.	A. & E.
		A. Vlei.	B. Vlei.	C. Vlei.	D. S. Ridge.	E. Vlei.	F. N. Ridge.			
	SPECIES.	Water Zone (*Sium-Equisetum*).	Silted Zone (*Equisetum* dominant).	Eastern Vlei Zone.	Sandy.	Western Broad Zone.	Clayey.	Frequency of Species.	Ob-served.	Critical Floristic Lists.
Cyperaceae........	*Bulbostylis complanata*, Linn...........	—	—	—	R.	—	—	—	SE.	—
	Carex cernua, Boalt....................	C.	—	L.	—	VC.	—	3 L.–C.	E.	C.
	Cyperus pulcher, Thunb................	—	—	L.	—	—	—	—	—	—
	Eleocharis limosa, Schultr..............	L.	—	C.	—	C.	—	—	SE.	—
	Fimbristylis complanata, Linn..........	—	—	C.	C.	—	—	—	SE.	—
	Fuirena pubescens, Kunth.............	—	—	R.	—	—	—	—	—	—
	Kyllinga elatior, Kunth................	—	—	—	—	L.	—	—	SE.	—
	Pycreus flavescens, Reichb..............	—	C.	C.	—	VC.	—	3 C.	E.	—
	Pycreus macranthus, C.B. Cl..........	—	—	R.	—	—	—	—	SE.	—
	Pycreus umbrosus, Nees...............	AD.	C.	A.	—	AD.	—	4 A.	RE.	AD.
	Scirpus corymbosus, Roth..............	A.	C.	C.	—	VC.	—	4 CA.	E.	VC.
Juncaceae.........	*Juncus exsertus*. Buch..................	—	L.	—	—	R.	—	—	SE.	—

Liliaceae	*Scilla minima*, Baker	—	—	L.	—	L.	—	—	—	—
Polygonaceae	*Polygonum lapathifolium*, Linn.	L.	—	—	—	L.	—	—	—	L.
Ranunculaceae	*Ranunculus pinnatus*, Poir.	C	—	—	—	VL	—	—	SE.	L.
Leguminosae	*Acacia robusta*, Burch.	R./RE.	R./RE.	—	—	—	R./RE.	3 R.	RE.	—
	Argyrolobium sp.	—	—	—	—	—	R.	—	—	—
	Indigofera woodii, Bolus	—	—	—	—	—	R.	—	—	—
Oxalidaceae	*Oxalis corniculata*, Linn.	—	L.	—	—	—	—	—	—	—
Umbelliferae	*Sium thunbergii*, DC.	C.	VL.	—	—	—	—	—	E.	—
Asclepiadaceae	*Asclepias fruticosa*, Linn.	R.	—	—	—	—	—	—	—	a.
Scrophulariaceae	*Nemesia parviflora*, Benth.	—	—	R.	R.	—	—	—	—	—
	Mimulus gracilis, R.Br.	R.	—	—	—	—	—	—	—	—
	Veronica anagallis, Linn.	R.	R.	—	—	L.	—	3 R.–L.	T.	R.
Plantaginaceae	*Plantago major*, Linn.	—	T.	—	—	—	—	—	—	—
Campanulaceae	*Lobelia erinus*, Linn.	—	—	R.	L.	—	—	—	—	—
Compositae	*Arctotis scaposa* (Harv.), O. Hoffm.	R.	—	L.	—	—	—	—	—	—
	Gerbera ambigua, Sch. Bip.	—	—	L.	—	—	—	—	T.	—
	Helichrysum aureo-nitens, Sch. Bip.	R.	R.	—	L.	—	R.	3 R.–L.	—	—
	Helichrysum caespititium, Sond.	—	—	—	C.	—	—	—	—	—
	Senecio erubescens, Ait.	R.	—	—	—	—	R.	—	—	—
	Senecio latifolius, DC.	L.	—	—	—	—	—	—	—	a.
	Sonchus nanus, Sond.	R.	—	—	—	—	—	—	E.	—

ABBREVIATIONS USED.

Prevalence of Species.
A = Abundant.
D = Dominant.
C = Common.
FC = Fairly common.
VC = Very common.
L = Little.
VL = Very little.
R = Rare.

Grazability of Species.
E = Eaten.
RE = Readily eaten.
SE = Slightly eaten.
T = Tasted.
a = Avoided.



Vlei Poisoning (Valley Poisoning).

Section VII.

Sterility.

J. Quinlan ... ... Researches into Sterility of Cows in South Africa.

*15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.*

Researches into Sterility of Cows in South Africa.

By PROFESSOR JOHN QUINLAN, F.R.C.V.S., Dr. Med. Vet., D.V.Sc.

PREFACE.

IN this work it is not intended to discuss in detail all the pathological conditions which are described in textbooks dealing with the diseases of the genital organs of cattle. The pathological changes described are confined to those which have been observed in South Africa during two years' investigations into the nature of sterility in cows. However, practically all conditions described by investigators in countries where intensive cattle breeding is carried out, such as America, Germany, Switzerland, Holland, and the British Isles, have been encountered. There is scarcely an important pathological change associated with sterility in cows which has not been met with. The work covers almost fully the whole question of the cause, symptoms, macroscopic and microscopic pathology, and the treatment of bovine sterility as it is observed in South Africa. The present work is the first publication in this country on the pathology and therapy of the diseases of the female bovine genitalia. The urgent necessity for such researches had been recognized within recent years, owing to the ever-increasing prevalence of sterility in bovine females. The opportunity for carrying out these researches was afforded me by Sir Arnold Theiler, the late Director of Veterinary Education and Research in South Africa, and his successor, Dr. P. J. du Toit, the present Director of Veterinary Services, to whom I wish to express my indebtedness for having put at my disposal an immense material for researches into a hitherto unstudied problem in this country.

This paper was completed in May, 1928.

Researches into Sterility of Cows in South Africa.

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J. QUINLAN.

INTRODUCTION.

During recent years the increase in sterility among cattle in this country has attained enormous economic importance. Since the introduction of imported cattle from Holland and the British Isles into the best known herds, the ever-increasing prevalence of sterility has come frequently to the notice of the veterinarian.

The importation of cattle and the subsequent grading of herds, which has taken place within the past fifteen years, have produced cattle which are very valuable from a breeding or milking point of view, but sterility, in many cases, reduces their value to what they are worth in the abattoir.

There is no doubt that a limited amount of sterility has always been existant among the native breeds of cattle in South Africa, but as it was of relatively little economic importance prior to the development of the pure-bred cattle industry, it was not brought so prominently to the notice of the veterinarian. At that time the expense connected with investigations into its causes, pathology, and therapy would scarcely be justified, except from a scientific standpoint. The result was that until recent years sterility and the many diseased conditions of the female genitalia associated with it received but scant attention.

There is little doubt that the hardier native cattle are more resistant to diseases of the genital tract than the pure-bred imported cattle. Further, it has taken years for imported cattle and their offspring to become really acclimatized to South African conditions, with the result that their resistance to disease was for a time at least lowered.

With regard to the prevalence of sterility in cattle, an attempt was made to ascertain the incidence in South Africa and Rhodesia by asking well-known farmers to answer a questionnaire. Prior to commencing our researches it was intended to compile statistics as to the percentage of farms on which sterility, either general or as isolated cases, existed. By general sterility in this Report is meant a farm on which over 20 per cent. of females of breeding age gave trouble in conception. The farmers were also requested to state whether contagious abortion or contagious granular vaginitis was known to exist. The following table is made up from information received from 215 replies. Letters which did not contain accurate information are not included. The number of cattle concerned would be approximately 10,000.

Union of South Africa.

Province.	No. of Herds Concerned.	Herds Affected.	Sterility.		Herds Affected with	
			General.	Isolated.	Contagious Abortion.	Contagious Granular Vaginitis.
		Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Cape	63	38·0	4·8	33·3	6	19
Natal	50	48·0	4·0	44·0	16	12
Transvaal	48	39·6	2·0	37·5	8	6
Orange Free State.	42	40·4	2·3	38·0	11·9	19
Total	203	41·5	3·27	38·2	10·47	14
Southern Rhodesia	12	41	8	33	8	16

From the above table the high incidence of sterility amongst South African cattle will be apparent. The percentages given regarding the herds infected with contagious abortion and contagious granular vaginitis are probably much too low. Neither disease is scheduled, so that accurate official statistics are unavailable.

The prevalence of the condition has undoubtedly greatly increased within the past fifteen years. This is due to the fact that cattle, especially the milking breeds, are now being kept under more intensive conditions; their environment simulates more that in Europe and America. It is a fact registered in other countries that sterility is more frequent in cattle which are kept under what must be considered artificial conditions of domestication. Housing with insufficient exercise, high feeding with the object of producing high milk records, control of breeding with the object of prolonging the lactation period and giving a high milk yield during the winter months, when milk is most expensive, are factors which must be considered artificial, and which tend to induce a deviation from the normal physiological activities of the female genitalia. These artificial conditions appear to upset that balance of the hormone secretions which recent literature (Marshall, Frei, and others) connects with the normal functioning of the genital tract.

The exact rôle of the internal secretions in the production and maintaining of functional sterility is not yet entirely understood, but the results which can be obtained in the treatment of cases of this nature by gland transplants and the use of ovarian extracts leave no doubt that the rôle is a not unimportant one. Further investigations, which are now being carried out along this line of research in other countries and at this Institution, will throw more light on this interesting subject. During these investigations the possibility of the hormone secreting glands, either individually or in groups, outside the ovary, being involved in the production of or associated with the pathology of sterility has not been lost sight of. In all cases of incurable sterility and in many cases of functional sterility, which did not yield to treatment, they have been systematically examined. It may be stated here that, although pathological lesions have been found in the thyroid, hypophysis, epiphysis, thymus, and adrenal glands in some cases, these lesions were not constant. It would, therefore, be unwise at the present time to associate them with sterility, either as a pathological consequence or an aetiological factor. Changes of the thyroid have been connected with sterility by several observers including Frei and Pugh. Struma colloides has occasionally been seen in sterile cows during these observations. This, however, would appear to be a common pathological lesion in the thyroid gland of other cattle in the Transvaal (Quinlan). Hyperaemia of the hypophysis has also been observed in some of the cows slaughtered. Here again the hyperaemia was not a constant condition in sterile cows suffering from the same pathological lesion in the genital tract. Other lesions found were corpora amylaceae in the epiphysis, calcareous deposit in the pars nervosa of the hypophysis, vesicles in the pars distalis of the hypophysis, apparent increase in the medullary areas of the thymus and increase in size of Hassal's corpuscles. In one case fatty degeneration of the adrenal cortex was present, in another there was fatty infiltration. In several of the adrenals there was irregular distribution of lipoid granules in the cortex. In one adrenal there was an encapsulated haemorrhage. No change was ever observed in the pancreas. The ductless glands from all animals slaughtered have been carefully measured and weighed. Comparisons have been made with the glands of normal cattle destroyed in the abattoir and also with the records obtainable from the works of such well-known authorities as Sisson and Ellenberger. The results have been practically identical, with the exception of the thyroid and adrenal glands, which showed a considerable increase in size and weight in the sterile cattle. This great difference can possibly be explained in the fact that, with one or two exceptions, all the cows destroyed for these investigations were pure-bred Frieslands, Shorthorns, or Herefords, which were much better grown and in infinitely better condition than the cross-bred cattle with which the comparison had to be made. It is intended to continue the study of the ductless glands in sterile cattle further. The study will be directed especially towards cattle which suffer from functional sterility, as it is in these that any pathological change is most likely to be manifest. Functional sterility can, in the majority of cases, be treated successfully by the careful selection of gland extracts, especially those obtained from the ovary. It is in these animals, therefore, that there is hope of obtaining definite results. At present it appears to be safe to state that the majority of cows which are sterile because of pathological lesions in the genitalia, such as metritis chronica catarrhalis or purulenta, hydrometra, salpingitis, and cystic degeneration of the Graafian follicle or corpus luteam, do not show any constant pathological change either on macroscopic or histological examination of the ductless glands outside the ovary. The changes which are evident in the physiological activities of the genital tract in these cows can as a rule be accounted for by a microscopic examination of the ovary. In the attached Appendix (No. V) a comparative table is given of the measurements and weights of the ductless glands in sterile and healthy animals.

The part played in the production of sterility by the variety and quantity of food given to breeding animals and also exercise has long been recognized (Zschokke and Dolder). Over-fat animals are known to be shy breeders. In these animals the oestral periods are few with long inter-ovulation periods. The condition, which is described as functional sterility, is most commonly met with in heifers, especially the beef breeds, which have been long maintained in high condition for show purposes. It, however, also occurs, but with less frequency, in heifers of the milk breeds when they are kept under similar conditions of inactivity and obesity. At this Institute, it has been the practice for years to import young heifers which act as virus reservoirs for red-water and gall-sickness vaccines. It is necessary to maintain these animals in high condition so that the maximum of blood can be withdrawn. As they must be maintained free from ticks exercise is restricted to a minimum. The food fed consists chiefly of maize meal, bran, lucerne hay, teff hay, maize ensilage, veld hay, and 1 ounce of salt daily. So as to ensure freedom from ticks, the ration is sterilized in bulk by steam.

The result of high feeding and restricted exercise is obesity. The heifers are used as reservoir animals for the production of redwater and gall-sickness vaccine until about the age of five years, when they are sold or sent to the Schools of Agriculture as breeding animals. Quite 80 per cent. of these heifers suffer from functional sterility of a very obstinate nature. The majority end in the abattoir as hopelessly sterile. Within the past two years those heifers have been used for experimental purposes at this Institution and several older reservoir heifers have been collected from the field for observation. The young heifers, as a rule, respond to suitable treatment with a balanced ration, compatible with high fertility in cattle, and the injection of ovarian hormones or gland grafts, but the older ones are very obstinate to these methods of treatment. However, many of them eventually breed. The first essential is to change the condition from one of obesity to that of physical fitness. At the moment it is too early to say whether having had one calf they will breed normally in future.

It is not certain to what this functional sterility can be ascribed, whether to changes in the secretion of the ductless-gland system or whether the change is not entirely confined in the ovary itself. It is certain, however, from our observations, that, in some old animals which have suffered from functional sterility since heiferhood, there is a change in the uterine mucosa and an atrophy of the cotyledons, so that there is a tendency for these animals to abort if they do conceive. In those cases the atrophy of the cotyledons and the mucosa may be due to disuse. Such atrophy of the cotyledons is however, commonly met with in cows suffering from metritis chronica catarrhalis and sterility for considerable periods of time. During the long inter-ovulation periods the ovaries also show an inactive state. They are small and firm. The corpus luteum becomes reduced in size and becomes brownish in colour. (Fig. 57, A. 2.) It seems to retain this state for weeks at a time. The other ovary shows no tendency to normally rapid development of Graafian follicles. Oestrum may occur at intervals of 2 to 3 months or at intervals of 4 to 5 weeks, or the oestral periods may be at normal intervals of 20 to 21 days in younger cattle.

The importance of the rôle played by a suitably balanced ration in breeding animals has been pointed out by many observers in other countries. Marshall, quoting Kirkham, points out that white mice are sterile after they have put on a large quantity of fat. Quoting Chalmers Watson, he states that rats are usually sterile when fed exclusively on meat. He has observed long inter-ovulation periods in very fat animals. Gibbons states that the ratio of barren to fruitful marriages in the obese is 1 to 5, whilst the ordinary ratio is 1 to 10. Gebhardt cited by Gibbons attributes the changes in the ovaries under these circumstances to changes in the thyroid and the adrenal glands. Experiments carried out by Marshall and Heape indicate that there is a greater percentage of twins and triplets in sheep fed on corn and turnips during winter than in those allowed to run at grass without concentrates. Further, intensive feeding caused the sheep to come into oestrum earlier than is usual. Graves and Miller have indicated the possible good results of vitamine E, as fed in sprouted oats, as a corrective in cases of functional sterility.

During the earlier stages of functional sterility a change in diet, coupled with normal exercise, in most cases produces the desired result. In the treatment of sterility a rule is made to put each animal on to a balanced ration containing crushed oats, bran, linseed cake, teff hay, green lucerne and grass. Bonemeal and salt are also given. This treatment has been successful after a varied period of time in allowing the normal oestrous cycle to appear in most cases of younger heifers suffering from functional sterility. Many heifers bred normally after the change of diet. It is, therefore, permissible to state that if high fertility is to be maintained, a suitable ration must be fed. Let it be understood that the heifers are first brought down in condition by exercise and a light ration.

It is not intended to discuss fully the literature on the subject of diet and sterility here. The question will be considered at a later date when our investigations into functional sterility are being discussed. It is considered sufficent at this juncture to indicate that in South Africa as well as in other countries the oestrous cycle and normal breeding is frequently upset by an unsuitable ration, especially when associated with insufficient exercise.

Underfeeding to a lesser extent also causes upset in the normal physiological changes in the ovaries. This is well marked in areas affected with drought in this country. Cattle in very low condition as a result of semi-starvation do not show a normal oestrous cycle. When extremely low, oestrous does not appear until the condition improves as a consequence of feeding concentrates or until the veld improves after rain.

Dolder states that anaphrodisia is common in under-nourished cattle and also in cattle fed continuously for high milk production. Richter draws attention to the relatively low fertility in badly nourished cattle, sheep, goats, and pigs during and following the war in Germany. Similar observations have been made by others in small animals. Gibbons draws attention to the low fertility in badly nourished women.

A chart showing the oestral periods of a number of sterile cattle is shown in Appendix IV. The diagnosis is given in each case so that it is possible to compare the oestrous cycle of cattle suffering from various conditions causing sterility with that of normal cattle.

It is necessary to draw attention to another important point regarding this so-called artificial environment. The diseased conditions which cause sterility other than functional sterility are in most circumstances due to some form of infection. It is not meant to convey the impression that sterility is solely due to contagious abortion and other contagious diseases. Contagious abortion, contagious granular vaginitis, and tuberculosis, no doubt, play an enormous rôle in the production of the pathological conditions resulting in sterility or low fertility, but there are other organisms which do not generally produce such conditions, but which may in certain individuals cause inflammatory changes of portions of the genital tract that also result in sterility. Housing and close concentration of cattle, feeding, milking, and shows, which bring cattle into close contact, predispose to the spread of contagious disease and the distribution of non-infectious disease of the genitalia causing sterility. By producing conditions favourable to the spread of disease intensive dairy farming has increased the prevalence of sterility. In this connection statistics show that the percentage of sterile cows amongst dairy cattle is much greater than amongst those of the beef breeds which are run under ranching conditions on the veld.

Neglect of stall hygiene is also a culpable factor in the conditions of domestication under which cattle are maintained. Badly lighted, badly ventilated, and unclean byres tend to lower the resistance of cows to infections, which such unhygienic premises undoubtedly harbour. One frequently encounters extensive outbreaks of puerperal metritis and cervicitis which can be easily explained in these byres. In fact, it would be difficult to understand how a cow could pass through the puerperal period without infection gaining entrance to the posterior genitals. The fact that a diseased stud bull may also be responsible for the increasing prevalence of sterility in South Africa must not be overlooked—certainly, however, to a lesser extent than diseased cows. In some herds where a low fertility record was shown in the absence of pathological lesions in the genital tract of the cows, the culpable factor was the bull. It is not intended to convey the impression that this occurs in nearly the same percentage of cases as in females, but it is brought to notice as a not negligible factor.

The neglect of sex hygiene cannot be overlooked as an important factor in the spread of diseased conditions causing sterility. How often do we see, even in well-bred and otherwise well-cared-for herds, a cow with a retained afterbirth standing in the byre at milking time? More often, however, one sees an animal with a purulent discharge from the genital tract contaminating its fellows. Cows suffering from sterility are bred, and as many of them come into oestrum far more frequently than normal, the bull stands the greatest risk of becoming infected and infections of the genitalia are conveyed to other members of the herd.

Of primary importance in the immediate cause of sterility is chronic inflammation of the uterus and the cervix. This fact was first established by Albrechtsen. He is supported by many well-known obstetricians at the present time, including Williams, Richter, Nielsen, Oppermann, and others. Our observations in South Africa confirm this view. The other lesions commonly found in the genitalia, such as salpingitis and pathological ovarian changes, are without doubt due to extension of infection from the uterus or the cervix. This fact is proved when one considers that changes in the fallopian tubes and the ovaries are less frequent than changes in the uterus in sterile cows. Further, salpingitis and cystic ovary rarely exist without a concurrent metritis chronica catarrhalis. The uterine change may not be clinically diagnosable, or it may not even be diagnosable macroscopically. However, microscopic examination, as a rule, reveals its presence.

Abnormalities in the oestrous cycle can be considered as a reflection of the state of the ovaries. Nymphomania and anaphrodisia are secondary symptoms of diseased conditions in the ovary and not in themselves a disease.

The diseases of the genitalia of cows produce sterility in a number of ways according to the pathological entity which is established in each case:—

(1) The spermatozoa or the ovum may be destroyed by exudates from an inflammatory lesion, and the development of spermalysin or spermatoxin. This may take place in the vagina, cervix, uterus, or fallopian tube.

(2) The ovum and spermatozoa may be prevented from coming together in cases of abnormalities in the fallopian tubes, uterus, cervix, or vagina.

(3) The fertilized ovum may be destroyed by bacterial toxins, or there may be death from malnutrition as a result of failure of the developing embryonic envelopes to become embedded in the degenerated mucosa of the cotyledons of the chronic metritic uterus.

(4) Static condition of the ovaries resulting in failure to ovulate in functional sterility, cystic degeneration of the Graafian follicle, or corpus luteum and corpus luteum persistens.

(5) Silent heat which occasionally occurs in cows and heifers, in which oestral symptoms do not occur although ovulation takes place.

(6) Deformities of the genitalia which prevent normal coitus.

(7) Arrest in the development of the female genital tract such as occurs in " freemartins."

DEFINITION.

Sterility may be defined in a general way as the inability of a female to bring forth living young. For the purpose of conveying the meaning as to what a sterile animal is during these experiments this definition is somewhat too general. The word sterility when used in these experiments means the failure of a sexually mature animal to conceive, or of the fertilized ovum to become implanted, after being served three or more times at different oestral periods by a bull of known high fertility, or the failure of a non-pregnant animal to show oestrum or ovulation.

CLASSIFICATION OF FEMALE BOVINE STERILITY.

General.

1. Congenital.
2. Acquired.
3. Relative or temporary.
4. Absolute or permanent.

In this case permanent sterility may be congenital or may have arisen after gestation has occurred. Temporary sterility is that in which the condition producing it can be overcome, whether it be due to anatomical, functional, or inflammatory causes.

Special.

1. *Anatomical.*
    - A. Vulvar.
    - B. Vaginal.
    - C. Cervical.

D. Uterine.
E. Tubal.
F. Ovarian.

2. *Aetiological.*
    (1) *Infectious Sterility caused by:*
        A. Tuberculosis.
        B. Contagious abortion.
        C. Contagious granular vaginitis.
        D. Non-specific infections.
            Diseases caused by these infections:
                a. Vulvitis.
                b. Vaginitis.
                c. Cervicitis.
                d. Metritis.
                e. Salpingitis.
                f. Ovarian changes.
    (2) *Non-infectious Sterility.*
        A. Congenital and acquired deformities of the genital tract.
        B. Functional sterility.

The discussion of the various diseases considered during these investigations will follow chiefly the classifications recorded under anatomical and aetiological.

METHOD OF INVESTIGATION.

Before commencing these researches, an intimate knowledge of the anatomy and histology of the female genital tract, the pathology and therapy of its diseases, as published in other countries, was gained by consulting the standard works of Sisson, Hess, Zschokke, Williams, Albrechtsen, Richter, Oppermann, Wester, Gordan, Joest, Aschoff, Kaufmann, Frei, and Ellenberger. Clinical experience and the value of therapeutic and preventive measures were gained by the examination and treatment of cows and heifers all over the country. For histological examination the genital organs and the ductless glands of sterile cows, which had been under observation for considerable periods at this Institution, were used. These animals were collected at the Laboratory from farmers who kept a record of the history of their animals, so that in most cases the previous history of each animal was known. They were selected after a careful gross examination of the genitalia as suitable subjects for our investigations. They were kept under close observation; their behaviour during oestrum and during the interovulation period was recorded. When considered desirable, the cows were slaughtered at the Pretoria Abattoir and the material collected immediately after death. Fresh specimens were cut and placed in 10 per cent. formalin. The genital organs were closely examined, measured, and described. The cross measurement of the cervix was taken about the junction of its middle and caudal third. Its length and width of wall were also recorded. The uterus body was measured from the cranial extremity of the cervix to the outward bifurcation of the horns. Its width was taken just in front of the cervix over the pars indivisa. The horns were measured from the bifurcation to the apex. Their width was taken about the junction of the middle and caudal third. The organs were then painted by the artist attached to the Institution. All the photographs of the genitalia appearing in Appendix II are taken from the artist's coloured paintings. The ductless glands were measured and weighed.

Sections from the various portions of the genitalia were cut and stained with haemalum and eosin, Van Giesen, and sudan III. When necessary, differential stains were employed. As time permitted the histological examinations were completed. The results obtained are included in Appendix 1.

DISEASES OF THE VULVA AND VAGINA.

Stenosis of the vagina has been frequently met with in herds in which treatment for infectious vaginitis has been carried out. As a rule, isolated cases of more or less marked stenosis were met with. The lumen of the vagina was decreased in size so that it admitted only one or two fingers. There was one herd, however, in which every cow which had been treated showed a complete or almost complete stenosis with consequent sterility. A dairyman maintained a herd of 70 pure-bred and grade Friesland cows of a really good type. About two years ago cows began to return to the bull. The condition became alarming as the owner had a milk contract in one of the largest towns in the Union. A qualified practitioner was consulted. He diagnosed infectious vaginitis as the cause of the trouble, without making a clinical examination of the genitalia of a single cow above the vulva. Neither was a serological test made for contagious abortion. He advised treatment of the whole herd. The owner was unable to carry out treatment of all the cattle at the same time. Forty-five cows were carefully irrigated with some drug supplied by the veterinarian. After irrigation, the cows showed extreme uneasiness and severe straining. In spite of this, treatment was continued for six weeks. The cows showed a purulent vulvar discharge from the commencement, and later it became impossible to insert the irrigator—a rubber tube—beyond a few inches. At the end of six months these cows were examined and all were found to be suffering from almost complete stenosis of the vagina. The lesion as a rule began at short distance in front of the urethral opening. Rectal examination showed that it extended forward in every case almost to the cervix. Some of the lesions were penetrable with a probe, but in no case could the little finger be passed. Those with complete stenosis showed the presence of liquid accumulation immediately posterior to the cervix. In those where the lumen was not entirely closed the anterior genitalia did not show any pathological change on palpation.

The cause of vaginal stenosis appears to be due to irrational treatment of infectious vaginitis or retained afterbirth by the use of strong irritant disinfectants. There are cases, however, where the lesion has been confined to the roof or floor of the vagina which could be associated with parturition injuries. Williams has indicated the frequency of vaginal stenosis when potassium permanganate has been used as an irrigant in bovines.

When stenosis is very marked so that the penis of the bull cannot penetrate the lumen, sterility results. In such a case the penis of the bull was severely injured in trying to copulate, while the cow suffered a severe laceration. When the lumen is open so as to admit two fingers, normal copulation and conception can take place. The danger of injury to the vagina and the penis of the bull in these cases cannot, however, be overlooked. In one case that came under observation, the vagina was badly stenosed, so that it admitted only two fingers with difficulty. The owner was recommended not to allow copulation, but when the cow was examined some months later she was found to be recently pregnant. Further, parturition followed without assistance and with only a relatively slight injury to the vagina.

There is another aspect of stenosis that cannot be overlooked when the lumen of the vagina is much decreased in size. The normal mucous and physiological secretions of oestrum sometimes accumulate in the pocket between the constriction and the cervix. It is not uncommon to find accumulations of a tenacious mucous material which appear to be the cause of sterility in occasional cases of stenosis. Clinical examination of the cervix and the genitalia anterior to it in these cases shows an apparently normal condition.

In general, it may be taken that stenosis of the vagina and vulva plays an unimportant rôle in the causation of sterility. The case described in which over 50 per cent. of the cows in the herd were sterile as a result of vaginal constriction must be considered exceptional.

Prophylactic measures should be considered when using vaginal irrigants. Strong or irritant disinfectants are contra-indicated for use in the vulva and vagina. Parturition injuries should receive surgical attention so that they will heal with the least possible degree of inflammatory phenomena and consequent restricted cicatricial formation. Unless stenosis is marked, it is unlikely to be a cause of trouble in copulation and conception. Neither is there a likelihood of dystokia. A postpartum examination, however, should be made to ascertain if the stenosed area has been lacerated. Cows suffering from marked stenosis should not be served, owing to the danger of laceration of the vagina and injuries to the penis of the bull. Attempts should be made to dilate the constriction and prevent recurrence by the application of boracic ointment, olive oil and iodoform, or some other suitable dressing. Mucous accumulations can be removed by irrigation with .9 per cent. saline solution. It may require repeated dilation before a cow is considered safe for service. When the stenosis is complete or almost complete, and the antero-posterior dimensions of the lesion are extensive, the prognosis is not hopeful.

Four cows have been met with in which there was a *thick band of tissue* covered with mucosa, which passed vertically across the portio-vagina of the cervix. The bands varied in width from 3 cm. to 6 cm., and in antero-posterior measurement from 3 cm. to 8 cm. All these animals had had several gestation periods and normal parturitions. The band was attached at either extremity to the roof and floor of the vagina. Its cranial surface was closely applied to the ostium uterinum externum over the caudal extremity of the cervix. It allowed the passage of two or three fingers between it and the cervix. These bands are due to remnants of the median walls of Müeller's ducts (Williams, Richter, Haesler, Hess, and others). It does not seem to interfere in any way with conception as a rule. This fact would point out that it is not necessary for the penis of the bull to penetrate the ostium uterinum for normal copulation; that it is sufficient insurance for conception if the spermatozoa are deposited in the anterior part of the vagina. In one case in which the band of tissue was somewhat T-shaped, 6 cm. across and 8 cm. antero-posteriorly, it would appear to have been the cause of sterility. The cow had bred normally previously. When examined, there was some ectropium of the last cervical fold so that the cervix was firmly pressed against the cranial surface of the band. The deformity was apparently the cause of sterility.

When these bands are found, they should be divided after first withdrawing the cervix to the vulvar opening. The ecraseur has been found a most useful instrument for division of those bands. The female ecraseur described by Korthaus was used with good results in the case described where the band was somewhat larger than is usually found. It is quite reasonable to conceive the possibility of their causing a delay in parturition should they pass between the limbs of a foetus, or the afterbirth may become entangled, preventing its expulsion (Williams).

Retention cysts in Bartholin's glands have been met with in six cases. The cysts varied in size from a pigeon's egg to an orange. There is no doubt that the cause is due to occlusion of the openings of the glands, due to inflammatory lesions in the vagina or vulva extending to the ducts. The cysts were oval or rounded in shape, confined to the lateral wall of the vestibule, about 8 to 10 cm. from the vulvar lips. They were firm in consistency. In all six cases they were unilateral. On section the cysts contain thick viscid slightly opaque contents. They do not play a part in the causation of sterility, as in the six cases in which they were found sterility could be explained in the presence of other pathological lesions. The cows in which the cysts were observed recovered, so that a histological examination of their structure was not possible. Rheinholdt described the histology as follows:—" Outwardly the cyst wall is covered with the mucosa of the vulva. Its epithelium is thickened. The wall consists of non-striated muscle mixed with more or less connective tissue, elastic fibres, and blood-vessels. The inner lining of the cyst wall is either composed of squamous epithelium on which the muscle fibres lie, or the epithelial lining is entirely absent, connective tissue only is present.

Surgical interference is not indicated unless there is evidence of enlargement which may cause difficulty in copulation or parturition and retention of secretion in the vagina. After exposure by opening the lips of the vulva, the cyst can be slit open and its contents pressed out. The lining can be freshened with a curette and painted with tincture of iodine. Healing occurs without difficulty.

In two cases *pathological changes in Gaertner's ducts* were seen. One showed the presence of cysts, the other showed abscess formation. The lesion consisted of several small spherical enlargements the size of a pea along the ventral aspect of the vagina. The contents were slightly opaque viscid fluid. In the case of abscess formation, there were three small abscesses somewhat larger than a pea containing yellowish thick-liquid pus. Williams states that these cysts may sometimes become very large. They are usually elongated, sometimes spherical. Frei says they may reach the size of a hen's egg. They appear to be of no importance in the causation of sterility. It is possible that they may become so enlarged as to interfere with copulation and cause retention of oestral secretions. When recognized they can be treated by incision and the walls painted with tincture of iodine.

Rupture of the dorsal wall of the vagina and penetration of the rectum was observed in one case (recto-vaginal fistula). The cow was a very valuable Friesland, 11 years old, which had given birth to a calf weighing 100 lb. about six months previously. She had since been served several times without result. On examination of the vagina, a considerable quantity of faeces was found accumulated in the anterior part. Two fingers could be passed into the rectum through a longitudinal slit in the dorsal wall of the vagina. When the rectum became distended the slit apparently opened and soft faeces passed into the vagina. The faeces which passed through the slit were partly expelled through the vulva and in part accumulated in the vagina. The cow's condition was poor, so that she appeared to suffer somewhat in general health from the lesion. The condition was a cause of sterility since no other pathological change was found at a clinical examination. The lesion would also constitute a grave danger to the penis of the bull during copulation. The cow made an excellent recovery after suitable treatment and again calved normally.

One case of *uro-vagina*, similar to the condition described by Richter, was observed in an old Friesland cow which had been sterile for a considerable time. On clinical examination the vulva admitted the hand with some difficulty. The vagina appeared to be dilated. There was an accumulation of about 100 c.c. of urine on the vaginal floor immediately behind the cervix. This seemed to be a constantly occurring condition. It is not possible to say whether the accumulation of urine caused sterility, since other pathological lesions were present which would account for a permanent inability to conceive. However, it would not be unreasonable to associate uro-vagina with sterility in exceptional cases.

Congenital hypoplasia of the genital tract would appear to be of some importance as a cause of sterility. It has been met with in six females out of a total of over 500 examined for sterility. The condition met with is usually referred to as "free martin," "neuter" or "queen." It occurs as a rule in the female of a mixed male and female twin pregnancy. In three of the cases under consideration the history of a twin pregnancy could be established, but in the remaining three an accurate history was unobtainable. Williams points out the possibility of a twin conception in which the male embryo perished in cases where a single birth can be definitely established. Richter says it is a condition which is found in nearly every case of mixed twin pregnancy in bovines. Keller found only 6 per cent. of females from a mixed twin pregnancy with a normal genital tract. Lüer found 5.3 per cent. from 113 cattle observed.

According to Zschokke, Keller, Lillie, and Zietzschmann, the deformity is due to the hormone secretion of the male twin influencing the further development of the genital tract of the female partner. The hormone reaches the female twin through anastomizing blood-vessels between the placentae of both foetuses.

In these animals one finds a small vulva which admits two or three fingers. The genital canal ends in a blind extremity a short distance from the vulvar lips. It is usually possible to find this *cul-de-sac* by introducing one hand in the rectum and the fingers of the other in the vulva. On a rectal examination, the cervix is found to be absent or undeveloped. The genital tract anterior to the vulva is undeveloped. Müeller's ducts can be felt as two parallel cords lying close together, or a partial fusion may have taken place. They may show some dilatation and contain some mucus. Two shorter cords, Wolffian ducts, can be felt on either side of Müeller's ducts. It is sometimes possible to penetrate into these through a very small opening from the *cul-de-sac* of the undeveloped vagina, but this can be done on post-mortem only. There is usually bifurcation at the anterior extremities of Müeller's ducts, representing the uterine horns, which may also be somewhat dilated. The ovaries are undeveloped. Small, indistinct glands may be palpated in the broad ligaments. (Fig. 14, 45B, A. 2.)

It appears that different stages towards more complete development have been described. It begins at the apices of Müeller's ducts where excavation takes place. This is followed by excavation of the uterine body after fusion of Müeller's ducts. Such a uterus may become filled with mucus; Williams states that this condition may be mistaken for pregnancy. Careful palpation, however, should obviate such a mistake. Where intimate examination through the vagina is not possible the animal can be re-examined. In the six cases which have come under notice a clinical examination revealed the condition without difficulty.

The general appearance is somewhat characteristic inasmuch as there is some coarseness. The refined appearance of the heifer is usually absent, but some neuters, however, do not show this characteristic. (Fig. 45A, A.2.) Oestral periods do not occur. In one of our cases a small corpus luteum was present in one of the ovaries. It is quite probable, therefore, that in some cases at least ovulation does take place.

These cattle with hypoplasia of the genitalia should be slaughtered when the first opportunity of the least economic loss presents itself. They are hopelessly sterile.

Richter mentions that the female partner of a mixed-twin pregnancy is as a rule destroyed in Germany, so that it is difficult to find an adult for examination. This practice is not adopted in this country; such animals are sold to breeders by auction without any guarantee whatever. The purchaser has no redress. The law is very lax in South Africa regarding the transfer of breeding animals suffering from a latent disease or condition which produces absolute sterility. Cases have come to our notice of cows having been sold by public auction where salpingitis was diagnosed a short time afterwards in the absence of an intervening pregnancy, or even oestral period. Since these cattle were sold without guarantee by the auctioneer, the purchaser has no redress. It is nothing short of fraud on the part of an owner to sell an animal to a breeder which does not conceive when bred to a known sound bull. It has, however, become a habit to " off load " such animals on to unsuspecting breeders at auction sales. With the present knowledge of the unlikely breeding capacity of the female of a mixed-twin pregnancy it is no less fraud to sell such an animal to a breeder. The fact that an animal is sold to a breeder is sufficient proof that he requires a female either in calf or capable of conceiving, and the seller should be responsible, in the absence of his expressing his willingness to submit his animals to veterinary examination, for reimbursement of the price paid and expenses incurred in transport and feeding.

THE AETIOLOGICAL SIGNIFICANCE OF INFECTIOUS VAGINITIS TO STERILITY.

The question of the aetiological significance of infectious vaginitis to sterility in cattle is one over which there is much controversy between veterinarians. The difference of opinion not only exists between veterinarians of different countries, but between the observers of each country. However, there is a tendency for the observers of each country to lean towards the same view. The majority of veterinarians in Switzerland and Holland believes that the disease plays an important part in the causation of sterility, while those of Denmark and America believe its economic significance in the breeding of cattle is negligible. Most observers, however, although they support the latter view admit that the question requires further investigation. They further recommend that under certain circumstances where sterility is present in a herd, in the absence of serological proof of contagious abortion and of pathological lesions in the ovaries, fallopian tubes, uterus, and cervix, where infectious granular vaginitis is definitely diagnosed it should be energetically treated.

The opinions of the highest authorities on obstetrics in countries where the disease has been studied within the last quarter of a century are interesting. Williams, who does not commit himself to a definite opinion, states:—

" It is perfectly clear that the nodular venereal disease has not been proven to be the direct cause of sterility or abortion in cattle or in other domesticated animals. Whether it does or does not directly cause sterility or abortion, clinical evidence indicates clearly that it is a disease which merits the respectful attention of investigator, practitioner, and breeder."

Nielsen states:—

" I am of opinion that it commonly causes sterility in heifers and that it is possibly the most frequent cause of the failure of heifers to conceive in spite of repeated services. Concerning this sterility in heifers, we are thus standing before an unsolved problem which richly deserves further investigation."

Albrechtsen, who must be considered as one of the leading authorities, states:—

" I have been unable to demonstrate the connection between follicular vaginitis and sterility "; and again: " I look upon follicular vaginitis as a non-malignant disease, which has nothing to do with sterility or, at least, is seldom associated with it, and the treatment of which is without influence on normal breeding."

Richter is of the same opinion as Albrechtsen, but he indicates the possibility of the disease having become less virulent since Hess published his opinion. It is rare, he states, to find an acute purulent infectious vaginitis in middle Germany such as was described by Hess 20 years ago.

Hess, who was one of the first observers to draw attention to the disease, states:—

"The disease is, in my opinion, and according to my experience and researches, of great significance as the cause of sterility in cattle. It appears as an enzootic or epizootic, and in some districts, on account of infection of the bull, is very prevalent. It appears in 75 per cent. of cases as a chronic condition and in 25 per cent. as an acute condition."

Hess further associated the disease with early abortions, from the 4th to 12th week of pregnancy. He states: "Another interesting fact is that cows 6, 9, 12 to 21 weeks after service show a purulent discharge from the vagina as a result of death of the foetus followed by maceration and endometritis chronica purulenta."

Zschokke states:—

"It has been proved that the disease may become the cause of sterility. Sometimes the bull loses the desire to copulate, sometimes cows and heifers will not allow copulation although they are in normal oestrus, or when conception follows, according to Obrecht, 20 to 60 per cent. of the cows abort."

Knell states:—

"I recognize the importance of other conditions, such as contagious abortion and pathological changes in the genital tract, which have been described by Hess. Often enough these conditions do not receive sufficient consideration. My observations, however, force me to consider infectious vaginitis as an independent infectious disease. In several cases, in the absence of all other causes of sterility, it delays conception or causes total sterility associated with repeated or permanent oestrum."

Martens states:—

"I have not observed infectious catarrh of the vagina as of undoubted aetiological significance in the cause of sterility."

Wester states that the disease as a cause of sterility has been exaggerated in Holland. However, he indicates that the exudate in acute inflammation of the vagina is toxic to spermatozoa, even after the acute symptoms have subsided.

Further quotations could be given from several veterinarians of high repute, but those mentioned above from several of the most reliable observers give an idea of the absolute different opinions held.

In South Africa, until recent years, the disease did not receive any attention from our profession. It was first recognized as being existant in 1912-13 in Natal. In 1920, owing to the prevalence of sterility in some of the pure-bred herds in Natal, the attention of the Division of Veterinary Research was brought to the question and advice sought. At that time most of the observations were carried out by a Swiss graduate, who perhaps followed the teaching of Hess. In certain herds where investigations were made and vaginitis was present it was inculpated as the cause of a high percentage of cows returning to the bull. In some herds which were treated for acute vaginitis, however, there was no history of sterility. Inquiries came from veterinarians in the Field Division as to the prevalence of infectious vaginitis and how it should be treated. It is feared that at the time treatment was undertaken in a somewhat promiscuous manner and that sufficient consideration was not given to other possible causes of sterility. It was taken for granted that if vaginitis was diagnosed in a herd, in the absence of serological proof of contagious abortion, that it was the cause of sterility. On this account, little value can be placed on the observations made at the time, and it is feared that the aetiological significance of the disease to sterility in this country has been very much exaggerated.

In considering the significance of the disease to breeders the following questions must be discussed:—

(1) Does infectious vaginitis play an important rôle in the causation of sterility in bovines?

(2) Should treatment be adopted to combat the disease?

These questions cannot be entirely discussed and conclusions drawn from the experience of other countries, as the prevailing climatic and other conditions may modify the disease; its virulency may be either exalted or attenuated. The disease must be considered as it occurs in South Africa. Further, there is sometimes reason to ask the question whether the purer breeds of cattle may not be more susceptible than the more resistant native and cross breeds, since it is not uncommon to find pure-bred cattle suffering from sterility while a grade herd on the same farm is entirely unaffected. When this latter question is carefully investigated it is found, however, that the conditions under which both herds are run are not exactly similar. In beef herds the grade herd is run under ranching conditions, while the pure-breds are housed and receive a supply of concentrates so that there is more intimate contact and thereby more danger of spread of infection. In milking herds where contact of the pure-bred and grade cattle occurs daily no such difference in susceptibility appears.

In 1901 Von Oestertag pointed out that the swollen follicles in the mucosa were not the characteristic symptom of the disease, but that the infectious nature of the discharge from the vulva was of primary importance. Zschokke was of the same opinion. Others, however, regard the swollen lymph follicles as a pathognomonic symptom of the disease.

Systematic clinical examination has shown that the diagnostic symptoms in the case of the acute condition are: Marked contagion and acute inflammatory lesions with swelling in the vulvar lips and mucosa, followed by swelling of the lymph follicles, muco-purulent exudate, sensitiveness of the mucosa vaginae, straining after copulation, and matting of the vulvar tuft of hairs.

The nodules due to hyperplasia of the normal lymph-follicles are not the chief clinical symptom through which the disease can be recognized. Several well-known observers do not place much importance on the presence of this follicular hyperplasia. They are certainly not of pathognomonic importance since they do occur in non-contagious forms of colpitis.

It is possible to differentiate between a peracute and acute stage, when the exudate is fairly profuse; a chronic stage follows in which there is little or no exudate and the follicles begin to disappear. The mucosa of the vulva and vagina becomes its normal pale pink colour. The transition to the chronic stage is sometimes fairly rapid, only a few weeks, sometimes it is very slow, taking several months. In the chronic stage the disease does not seem to be infectious. Martens says the infection is absent long before the swollen follicles have disappeared.

Infectious vaginitis was at one time thought to be a most fertile cause of abortion; as already explained, this was the belief of Hess. At the present time no veterinarian who has carried out careful observations holds this opinion. Williams, Wester, Albrechtsen, Richter and others have published observations which are sufficiently convincing to contradict this view. There is no doubt that the bacillus of Bang is the chief cause of abortion in bovines. Further, since the work of Hess and Albrechtsen was published the prevalence of disease of the ovary, fallopian tubes, uterus, and cervix and the part they play in the causation of sterility is known. It may be argued that these diseases are secondary to vaginitis, but the absence of the metritis of abortion, cervicitis, and metritis due to other organisms, not recognized as infectious, must first be proved before infectious vaginitis can be inculpated. It is not maintained that Bang's bacillus will cause pathological lesions associated with sterility. In fact, a uterus may undergo complete involution and be free from *B. abortus* in three weeks after parturition. It is unusual to find infection by this organism present six weeks post-partum. Early abortion and the frequency of placental retention, however, do open the portals for secondary infection, so that contagious abortion must be looked upon as the chief predisposing cause to the pathological lesions associated with sterility.

The question arises, if infectious vaginitis does cause sterility or prevention of conception, how is this brought about? The literature on the subject is far from convincing.

Schunhoff believes that it is due to the cramp-like closure of the ostium uterinum externum, due to reflex action from irritation of the vagina.

Ellinger believes that the irritation of the vagina causes too early expulsion of the semen through straining. This would seem unlikely, since it would be quite impossible to expel all the spermatozoa from the anterior extremity of the vagina, even assuming that none have entered the orificium externum.

Pfeiler maintains that infectious vaginitis, as such, is not responsible for sterility, but that the endocrine system associated with conception and pregnancy is influenced by the disease.

Wester and Knell maintain that the spermatozoa are destroyed by the unphysiological vaginal exudate of infectious vaginitis before they can reach the fallopian tube. Wester has shown the presence of spermatoxin in the exudate.

It is well known that spermatozoa die quickly in the normal vaginal secretion in vitro. They live longer in physiological saline. Much interesting work has been carried out by Wester on the toxicity of various chemical substances and body liquids on spermatozoa. He indicates that the exudate from the inflamed vaginal mucosa is toxic.

Schlichte took the spermatozoa of a young strong two-year-old bull, whose fertility could not be questioned, from the vagina of a cow after service. He mixed the spermatozoa with the vaginal secretion of a normal cow, with physiological saline and with the vaginal exudate of a cow suffering from acute infectious vaginitis. The spermatozoa mixed with secretion from a normal cow lived ½—1½ hours, that in physiological saline showed movement after 3 hours, that mixed with the vaginal exudate of the cow with vaginitis lived only ½ an hour. Wester carried out similar experiments with the same result.

Whether the same result takes place *in vivo* would require proof. However, there would appear to be something present in the pathological mucous exudate of infectious vaginitis which is toxic to spermatozoa in vitro.

The view held by Wester and Knell is at the present time that universally accepted by those who believe that infectious vaginitis plays an important part in the causation of sterility.

The disease is widespread throughout South Africa, especially in milking herds, where the facilities for the spread of disease, such as close housing and intimate contact, are more evident. However, in Natal it does not spare the beef breeds. It is believed by veterinarians and stockowners to be the main cause of sterility in bovines in that province. This view, however, would not appear to be upheld when the cause of sterility is carefully investigated. Many of the herds in Natal, where complaints regarding sterility and cows returning to the bull had been brought to the notice of the Research Division, were visited recently. With few exceptions, the sterility could easily be explained in the presence of abortion, or pathological lesions in the genitalia associated with permanent or temporary sterility. These pathological conditions cannot be regarded as secondary to vaginitis, but mainly to the metritis of abortion. In some cases, where 30 to 40 per cent. of the cows were sterile or returning to the bull, the condition could be explained as functional sterility due to errors in feeding, with insufficient exercise, etc. The sufferers were, without exception, cattle which had been maintained in high condition for "show" purposes and which got little exercise. It must be understood that a ration too rich in carbohydrates and low in vitamin, protein, and phosphates is not associated with high fertility; while such a ration in the presence of insufficient exercise produces obesity, a condition absolutely incompatible with conception. The state of low fertility in such animals is probably associated with upset of the hormone secretion. What endocrine glands are involved is not known. That the hormones secreted in the ovary itself are inabundant appears to be without doubt, as proved by the results obtained in these cases by the injection of oestral hormone (Murphy, McNutt, Zupp and Aitken; Frank and Goldberger; McNutt; Murphy; Marshall; Frank, Kingery and Gustavson; Zupp and Murphy). Good results have been obtained at this Institute in a number of cases treated with ovarian extracts. These results will be published when sufficient data are available. Some workers suggest that a combination of the endocrine glands, not the ovary alone, are hyposecretive. Pathological examination, however, shows that the ovary is as a rule inactive. Cattle in low condition as a result of insufficient food also suffer from oestral abeyance and temporary sterility. Richter has recently made interesting observations in this connection. No doubt the explanation of unbalanced hormone secretion also holds good in this case.

There were many cases where irrational treatment had been undertaken; treatment which would be prohibitive to conception in the absence of disease of the genitalia. In some of these instances no evidence of vaginitis in a culpable form could be found. The genitalia of the cows were normal. The drastic treatment, which was faithfully continued, was the sole cause of sterility. It has been pointed out that spermatozoa do not long survive in the normal vaginal secretion. It is therefore unlikely, as Wester demonstrated, that they will survive strong irritant disinfectants even for a few minutes. To illustrate the drastic and barbarous methods used in the treatment of infectious vaginitis, one example can be given. Reference has already been made to this case when stenosis of the vagina was being discussed. In a herd of 70 pure-bred and grade Frieslands, 45 of which had been treated for infectious vaginitis, all were found to be suffering from stenosis of the vagina sufficient to cause sterility. This was due to the effects of some irritant disinfectant which had been recommended by a veterinarian as an irrigant for the vagina. Treatment caused sloughing of the vaginal mucosa, which was followed by cicatrization and consequent stenosis.

The batch of cows left untreated certainly showed lesions associated with the chronic stage of infectious vaginitis. However, most of them were in calf or had calved within the previous six months.

This is not the only case in which irrational treatment has been observed during our investigations into the cause of sterility, but it is the worst case. The question arises, if such a state of affairs can follow treatment conducted under supervision of a qualified veterinarian, what is the position in many herds where veterinary advice was not sought? The farmer diagnoses infectious vaginitis himself and treats it with that amount of vigour which is characteristic of laymen's methods. For instance, it is quite a common practice amongst farmers to irrigate the vagina of a cow with a disinfectant a short time prior to service. Even when irrigation with light and carefully measured antiseptic fluids is carried out prior to copulation it will most likely prevent conception. Therefore, it is reasonable to suppose that irrigation prior to service has done unlimited harm in the hands of laymen. There is another danger in the practice of irrigation which cannot be overlooked; that is, the possibility of transmitting disease from cows suffering from purulent genital affections to healthy individuals.

It is a common belief that acidity of the vaginal secretion is frequently met with in cows. A rough examination with litmus paper would seem to indicate that this may be the case. Such a condition is associated with sterility not only by laymen but also veterinarians (Pusch-Hansen). As a treatment, irrigation of the vagina with a weak solution of sodium-bicarbonate is recommended, and success has been reported in some cases. Recent investigations into the reaction of the vaginal secretion by Kaden, Renkert, and Wester have proved this acidity of the vagina to be of rare occurrence. Richter states that acidity of the vaginal secretion plays no practical rôle in the causation of sterility. He points out that the apparently good results obtained by irrigation with bicarbonate solution are due to mechanical removal of tenacious mucus which prevents the forward passage of spermatozoa through the cervix.

In connection with the promiscuous treatment of infectious vaginitis, a quotation from Martens, who formerly believed that vaginitis even in the chronic nodular form was a fertile cause of sterility, and who carried out propaganda for a general treatment, is interesting. He states: "One is often astonished at the obstinate, useless, and partly barbaric fight against the mostly harmless nodules in the vulva."

Herds, however, have been met with in which infectious vaginitis was acute and widespread in company with frequent returning to the bull and sterility. This was specially noticeable in heifers and young cows. From these herds contagious abortion was excluded by serological tests. A careful clinical examination failed to reveal any pathological changes in the genital tract or ovaries except vaginitis. The fact that the bull may be an important factor in sterility was not neglected. The ration feed in these herds was well balanced and the condition of the animals could be associated with high fertility. Exercise was sufficient and hygiene good. Rational treatment of the disease soon overcame the prevailing temporary sterility. In such cases acute vaginitis certainly does play an important part in the causation of sterility. There is no doubt, as indicated by Williams, that irrational treatment of the disease and neglect of sex hygiene cause a marked increase in the intensity of the symptoms. Under these circumstances an extension of inflammatory phenomena to the anterior portion of the vagina and the pars vagina of the cervix may occur. In South Africa, in the absence of proper sex hygiene and the presence of irrational methods of treatment, one finds cervicitis so frequently associated with infectious vaginitis in sterile cattle that it would be unreasonble to assume otherwise than that cervicitis is a sequel. In the presence of cervicitis, as a sequel to irrational treatment of infectious vaginitis, it is not unreasonable to expect a sterile animal, since cervicitis caused by other infections is a most frequent cause of sterility. It is admitted that these arguments are circumstantial, but in herds where it was possible to exclude contagious abortion by serological methods, and where infectious vaginitis was intense, no other explanation could be given as to the cause of sterility. To substantiate the seriousness of the disease as a cause of sterility, attention may again be drawn to the pathological changes which the acute disease is capable of producing in the mucosa of the vulva and the vagina in cows and the sheath and prepuce in bulls. In many acute cases great irritation and straining follows copulation. The secretion of the mucosa of the posterior genitals under such circumstances cannot be otherwise than pathological. It is not unreasonable to assume, therefore, that the physiological functions of the copulatory portions of the genitalia are disturbed and temporary sterility results. Contagious vaginitis in the chronic or dormant form, in the absence of secondary lesions, would not appear to be a cause of sterility as its presence is compatible with high fertility and pregnancy.

From a review of the literature from other countries and experiences of the disease in South Africa, the following conclusions are justified:—

(1) Infectious vaginitis is an infectious disease which may exist independently of contagious abortion.
(2) Vaginitis as a causative factor in sterility has been greatly exaggerated in South Africa.
(3) Vaginitis in an acute form, which is most usually associated with bad stall hygiene, carelessness in sex hygiene, and irrational treatment, may cause temporary sterility and frequent returning to the bull, especially in heifers and young bulls.
(4) That in most herds where a more or less general (10 per cent. to 80 per cent.) sterility exists, other factors, such as contagious abortion, functional sterility, and pathological lesions in the genital tract or ovaries, can be proved.
(5) General treatment is indicated only after a careful clinical and serological examination has been undertaken by a veterinarian.
(6) Treatment is indicated when infectious vaginitis is acute, with a muco-purulent discharge from the vulva and when there is difficulty in conception in cows and heifers.
(7) Treatment should be undertaken only in company with the necessary measure of precautions against other diseases causing sterility which may exist concurrently.
(8) Treatment is not indicated in:—
    (*a*) The absence of sterility or difficult conception;
    (*b*) Pregnant cows.

DISEASES OF THE CERVIX UTERI AND UTERUS.

(*a*) DISEASES OF THE CERVIX UTERI.

PATHOLOGICAL CHANGES in the cervix which can be associated with sterility are of relatively frequent occurrence. *Anatomical deformities* observed may be of a congenital or acquired nature. They are probably most frequently congenital. Congenital and acquired pathological lesions have been described by all workers who have busied themselves with investigations into the pathological lesions of the genital tract in bovines, including Frei, Oppermann, Williams, Albrechtsen, Richter, Hess, Zschokke, Nielsen, and others. The bands of tissue which cross the portio-vaginalis uteri of the cervix due to persistence of the median wall of Mueller's ducts have been mentioned under diseases of the vulva and vagina. *Cervix duplex or double cervix* has been observed in one case. (Fig. 33, A. 2.) The subject was an aged Friesland cow which had several pregnancies and normal parturitions. The uterus was of the normal bovine type. The circumference of the cervix was much increased in size. The body was tapering towards the uterine extremity. The lumina of the double cervix were separated by a thick band of normal cervical tissue, which gradually decreased in thickness towards its cranial extremity. The ostium uterinum on the right side was of greater calibre than that on the left, but the latter admitted a thin sound without difficulty. The condition did not interfere with conception nor was there any difficulty at parturition. It would appear, however, that parturition difficulties might supervene should both ostia become dilated and the extremities of a foetus enter both cervices. Such difficulty could easily be overcome, however, by repelling the offending member or by division of the septum. No case can be found in the literature of dystokia from this cause. It is quite likely, however, that the one cervix only becomes dilated during labour. Williams draws attention to the fact that the condition may be congenital and be perpetuated or accentuated in the offspring.

Two cases have been observed in which there was a marked local *dilatation of the lumen of the ostium uterinum.* (Fig. 77, 44B, A. 2.) In one case there were two large compartments between the third and fourth and between the second and third annular folds. The wall was dilated and thin. In the second case there was a single dilatation between the second and third fold. These compartments were filled with a tough tenacious grey material, resembling the cervical seal of pregnancy, but somewhat softer. The lumen of the ostium uterinum through the cervical folds limiting the dilatations was reduced in calibre. The accumulation of cervical secretion had caused occlusion of the cervical canal and in both cases caused retention of the normal uterine secretions and oestral débris. Both cases were heifers which had never conceived and one of which had not been seen to be in oestrum. There is no doubt that at some period prior to purchase by this Institute that each had ovulated at least once, since there was a well-developed corpus luteum present. While under observation one animal showed a single oestrum. It would appear that the cervical condition caused occlusion of the cervix which resulted in a marked hydrometra in one case and retention of oestral débris and uterine secretion in the other. This was followed as a consequence by retained corpus luteum and the development of a false uterine seal. The cervical deformity in one case was associated with a uterus unicornis.

Stenosis of the cervix in heifers, described by Richter and Albrechtsen, has not been observed. An acquired complete closure of the anterior extremity has been met with. (Fig. 58, A. 2.) The subject was a Hereford cow which had had one normal parturition. It would appear that the lesion was probably due to a parturition injury. The cranial extremity of the cervical canal was entirely closed by a thick band of tissue. The intercornual septum was attached to this band of tissue, thereby obliterating the pars indivisa of the uterus. There was no communication between the uterus and vagina. There was a mucous accumulation in the uterus and a corpus luteum persistens in the left ovary. Williams, under the heading "Retention of menstrual débris from cervicitis," points out the extremely rare occasions on which this occurs. He has had only two cases of cervical stenosis or deformity causing retention, in his tremendous material.

Referring to stenosis of the cervical canal in cattle, Williams points out that it must be of infrequent occurrence. Observations in this country corroborate Williams' opinion. In all cases where atresia of the os was certain, the uterus was distended with oestral debris and a retained corpus luteum was present.

Cases of stenosis of the anterior portion of the cervical canal have been met with in two very old cows which had apparently suffered from chronic cervicitis. The ostium uterinum was so small that it admitted only a very small probe in its cranial third. The mucosa of the canal was the site of small cystic degeneration. The cervical wall was markedly sclerotic and inelastic. Further, pathological changes were present in the uterus and ovaries which could be sufficient to account for the sterile state of these animals, so it is not possible to state what part the stenosis of the cervix played. There was little mucous accumulation in the uterus. It, therefore, appears likely that the cervix was capable of sufficient dilatation during the oestral periods to allow of the discharge of physiological oestral secretions.

Another interesting deformity of the cervix observed was one of *lateral curvature*. The subject was a Red Poll heifer, three years old. The cervix was small in circumference and bent almost U-shaped towards the right. The remaining portion of the genital tract was normal on rectal palpation. Intimate vaginal inspection of the cervix was not possible. The heifer was kept under observation for a period of 12 months and during this period never showed oestrum.

Cervicitis.

This is a condition of inflammation of the cervix which usually exists in conjunction with metritis or as an independent condition. It is a disease of the utmost importance as a cause of sterility. The sterility which it produces may be of a temporary nature at first, in so far as most diseases of cervix can be successfully treated. Neglect of early treatment, however, results in many cases of incurable sterility as a result of subsequent invasion of the uterus and fallopian tubes.

Cervicitis is a result of bacterial infections of the mucosa of the cervix. These infections may reach the cervix in many ways. Williams points out the frequency of cervicitis in heifers, and refers the date of invasion of the cervix to calfhood. The cervix is not infrequently somewhat torn during parturition. Bacteria may easily gain entrance to these abrasions and remain there. The chances of infection are naturally more exaggerated when the afterbirth is retained. Williams states that the organism most commonly found in inflammatory lesions in the cervix is streptococcus viridans. There is no doubt that many other organisms may be responsible. According to Williams, the bull is probably a most frequent offender in the transmission of bacterial infections which cause cervicitis, either by direct mechanical transmission from diseased cows, or by transmission from a diseased male genital tract. He gives instances of widespread cervicitis, involving 60 per cent. of the cows in a herd, which were directly referable to diseased bulls. It frequently exists with a metritis, of which there is no doubt it is a most common sequel. It is not infrequent to find cervicitis in heifers which have not been served. Most frequently, however, it is associated with the presence of contagious granular vaginitis, or neglect of sex hygiene. It is not maintained that cervicitis is a direct sequel to extension of vaginitis forward to the cervix. It is found in association with acute vaginitis in herds where infectious vaginitis has been irrationally treated with irritant disinfectants and where no proper sex hygiene is carried out. In some herds, where 50 to 60 per cent. of the females were sterile, cervicitis has been found co-existant with infectious vaginitis. It would, therefore, be unreasonable to assume otherwise than that it may be a sequel. Another frequent cause of transmission of these infectious is neglect of proper aseptic precautions during the treatment of genital diseases: sterility, contagious granular vaginitis, metritis, and retained afterbirth.

Thorough examination of the cervix presents some difficulty. This is especially marked in the case of heifers when the hand cannot be introduced into the posterior genital passages. In such cases Albrechtsen and Oppermann advise incision of the vulvar lips to admit the hand. Even then it is sometimes extremely difficult to withdraw the cervix sufficiently for careful examination on account of the shortness of the broad ligaments in the heifer. The gripping of the pars vagina with the retraction forceps is usually possible when the second hand directs operations from the rectum. When examination cannot be carefully carried out and the cervical canal cannot be opened, the examination should be postponed until an oestral period when the canal is much dilated. Suitable instruments for the purpose are an absolute necessity. Two cervical forceps are required. For treatment and examination of the os uterus a cervical dilator, a small uterine canula, and a dressing forceps are also necessary.

Various degrees of inflammatory changes may exist in the cervical mucosa and the cervical wall. An acute and chronic inflammation can be recognized. In the acute stage the mucosa is always reddish to dark reddish in colour. It is swollen to a varying degree. In the less acute cases the first cervical fold is plainly visible. In acute cases the fold is protruding into the vagina, markedly tumefied, dark red in colour and deeply furrowed. The second fold is also usually involved and can either by seen or felt through the ostium uterinum. The ectropic folds may reach a diameter of several centimetres. Retraction of the acutely inflamed cervix easily produces haemorrhage. On rectal examination, the cervix is found swollen and somewhat increased in diameter, especially its caudal extremity. There is also an abnormal secretion from the cervical mucosa, varying from a thick tenacious mucus with some purulent contents to thin or thick liquid pus. The quantity of exudate also varies with the intensity of the inflammation. The tough tenacious material mixed with purulent flocculi accumulates in the anterior extremity of the vagina. It is usually small in amount. It sometimes hangs in thick strings from the vulvar opening and soils the tail. The pus accumulates in the vagina as a yellow thick, usually foetid, material which occasionally issues from the vulva and soils the tail and thighs. Both varieties of exudate are incompatible with conception, and animals suffering from cervicitis are as a rule sterile. The inflammation may be confined to the posterior portion of the cervix or it may involve the whole structure to its cranial opening. If the condition is allowed to exist, the inflammation passes into the deeper tissues in the wall, where it produces changes which cause a permanent hypertrophy of the cervix, or even stenosis and closure of the ostium uterinum. The mucosa in these chronic cases may assume a pale pink appearance. The cervical canal may be open throughout so that it admits the finger, or it may present local stenosis or complete stenosis as a result of hypertrophy and induration of the annular folds. The ectropic caudal fold is usually very ragged, irregularly and deeply furrowed. Polypus-like projections are also met with. The wall is abnormally fibrous, firm, and thickened. Further, the changes in the mucosa may be so that, should conception take place, it is unsafe and early abortion may result (Williams). Abortion in these cases is due to an endo-metritis accompanying cervicitis or to the failure of the pathological cervical mucosa to secrete the normal uterine seal of pregnancy, so that proper sealing of the pregnant uterus does not follow conception.

Little has been written regarding the micro-pathological changes of the bovine cervix. Suvanto described the histological changes in cases of cervicitis, and his observations are similar to those found during investigations at this Institute. In the acute cases there is epithelial desquamation and degeneration on the summit of the longitudinal folds of the mucosa. The bottom of the crypts may still show normal cylindrical epithelium. In the very acute cases there is desquamation of the epithelial covering in the depth of the crypt. The epithelium is covered with exudate mixed with some cell débris. The mucosa shows infiltration with neutrophiles, lymphocytes, and sometimes eosinophiles. The capillaries are dilated. In chronic cases the epithelium may show a normal cylindrical single layer of cells, or in parts a stratification three or four cells high. One case was observed in which there was adhesion between the mucous folds at their summits while the epithelium at the base of the crypt remained normal, so that there was a cyst-like formation in several sections examined. Richter has also described this condition. Eosinophilic infiltration can be observed in the mucosa in some sections. There is increase in round cells in the mucosa, showing that the process is active. There is marked increase in the connective tissue of the wall with local reduction or complete absence of non-striated muscle fibres. Occasionally large cells have been encountered having a finely granular acid staining cytoplasm and a round or slightly oval nucleus.

Cystic degeneration of the mucosa is occasionally met with in conjunction with cystic degeneration of the uterine mucosa. Macroscopically the cysts may be numerous or few in number. They are usually small, but some may be the size of a pea. They are most frequently seen in the cranial third of the canal, but may also occur throughout its entire length. They appear as rounded elevations with a semi-transparent or opaque capsule. On section they are smooth-walled and contain an opaque fluid. Microscopically they appear as simple cysts with a single layer of cubical or flattened cells surrounded by a fibrous tissue capsule. Marked cervical sclerosis has been concurrent with cystic degeneration. It is probable that these are retention cysts, the result of old-standing inflammatory lesions of the cervix. Such a pathological condition when widespread would most likely be of considerable importance in preventing conception. It is unlikely that the cervical mucosa is capable of normal physiological secretion. The development of the cervical seal of pregnancy would be interfered with. The portals to the uterine cavity remain open to infection, and early destruction of the embryo is probable should conception follow service.

The prognosis of cervicitis *per se* may be considered good as a rule. However, it depends on the acuteness of the condition and the stage at which it is treated. Williams states: "if intense and neglected, the infection tends to invade the oviducts and pass beyond surgical control." In those cases, however, it appears that the uterus acts as a connecting link and it is first involved in the extension of the inflammatory process. Cases of long standing are always doubtful, as it is not unusual to find that metritis and salpingitis are co-existant. In most of these cases one must look upon metritis as the primary lesion to which cervicitis and salpingitis are sequelae. Further, the cervical mucosa may have become so changed that it can no longer perform the physiological functions compatible with conception and pregnancy. It is highly probable that the inflammatory exudate is toxic for the spermatozoa, and that the tenacious mucous or purulent exudate also occludes the cervical canal. Light catarrhal cervicitis frequently passes off without treatment, the mucosa again returning to normal. Williams points out that there is great danger to the embryo after conception in cases where the spermatozoa pass the inflamed barrier. The canal does not become physiologically sealed and the infection is free to pass into the uterus.

The treatment of cervicitis again brings up the necessity for careful sex hygiene. Wounds of the cervix, placental retention, and puerperal metritis should receive careful attention. A cow which is suffering from a discharge from the genital tract during the post-puerperal period should not be served until it has been definitely ascertained what is wrong and the condition successfully treated. There is a real danger in transmission of disease to healthy individuals through the agency of the bull. It is, therefore, necessary to hold affected cows back from the bull and isolate them during treatment. In order to carry out treatment effectively, it is necessary to withdraw the cervix with a cervical retraction forceps. It may also be necessary, in some cases, to dilate the os uterus slightly with a dilator. The passage of a dilator through the os uterus requires the utmost care. It is necessary to follow the os slowly and without pressure, as rupture of the mucous membrane increases the danger by allowing organisms to pass more deeply into its wall. The danger of passing a dilator is all the greater when inflammation of the cervix is severe, as the wall of the os uterus is likely to be the seat of much pathological changes from ectropium, swelling, and hypertrophy. Having ascertained that the os uterus is patent and sufficiently dilated to allow of dressing, it should be cleaned out with a cotton wool swab, and then thoroughly swabbed throughout its entire length with tincture of iodine, "lugol's iodine," rivanol, or yatren solution. The swabbing can be easily accomplished by catching hold of a suitable-sized piece of cotton wool in the jaws of a cervical dressing forceps, rolling it carefully around the forceps to prevent injury to the cervix, and saturating it with lugol's iodine, or one of the other solutions mentioned. The forceps should be introduced deeply into the cervix (7-9 cm.) and every portion of its wall touched with the solution. This treatment can be carried out once or twice weekly while necessary. It has been pointed out that cervicitis is frequently a complication of other pathological changes in the genital tract, such as acute and chronic metritis, wounds of the cervix, retained afterbirth, and contagious granular vaginitis. It is unnecessary to mention that co-existing diseases, to which cervicitis is often a sequel, must also receive suitable treatment.

Williams, Richter, Oppermann, and others recommend the surgical removal of enlarged portions of ectropic annular folds or polypus-like growths. Such procedure would appear to be indicated in exceptional cases where the swelling causes occlusion of the canal. Polypus-like growths and enlarged annular folds are very frequently met with in aged cows after many parturitions and are compatible with pregnancy. When removal of enlarged portions of folds is attempted, uniting the mucosa with catgut sutures after deep removal, as described by Williams, appears to be a better method of procedure than that described by Richter, who removes superfluous tissue with the curette. Richter finds the circular amputation of the cervix described by Williams unnecessary. The operation has not been performed during these investigations. Nielson believes that vaginitis *per se* does not require any special local treatment. He indicates that the condition is cured by suitable attention to the acute and chronic metritis to which it is a sequel. Attention is drawn to the fact that tumefaction and hyperaemia of the cervix, which rapidly occurs during handling, must not be confounded with cervicitis.

(*b*) DISEASES OF THE UTERUS.

(1) Congenital and Acquired Deformity.

Deformity of the uterus plays only a minor part in the causation of sterility in cattle. Outside of "free-martinism," two cases of uterine deformity only have been met with.

One was a case of uterus unicornis in a Short-horn heifer which had never conceived. (Fig. 44 B, A. 2.) Williams has encountered this condition not infrequently. In abattoir specimens he has seen uterus unicornis six times out of somewhat more than 3,000 females examined. Five of these had been pregnant, while one was a heifer. It is, therefore, quite compatible with conception.

One case observed at this Institute was easily diagnosed per rectum. The uterine deformity was accompanied by cervical deformity which has already been referred to. (Fig. 33B, A.2.)

The horn which was present was well developed, but it was the site of a hydrometra from retention of oestral débris. There was no evidence of the normal cotyledons of the non-pregnant uterus on the mucosa. The retention of débris was due to cervical deformity and the development of a false uterine seal. The fallopian tube on the side of the absent horn ended in a *cul-de-sac* towards the extremity distal to the ovary.

The second case (Fig. 58, A.2) was an acquired deformity, apparently the result of a severe injury to the cervix during parturition. The cervical canal was shortened towards its cranial aspect. The orificium internum was completely occluded. The intercornual septum and the cranial aspect of the cervix were closely adherent through thick connective tissue adhesions. The pars indivisa of the uterus was not evident. A uterus bipartitus completus had been established. The horns contained a small quantity of somewhat tenacious clear mucus. There was no marked distension of the uterus with oestral débris or mucous exudate although the occlusion of the ostium uterinum existed many months.

(2) Endometritis Chronica.

Chronic inflammation of the uterus is by far the most common lesion met with in cows suffering from sterility. It is without doubt the cause of sterility in 70-80 per cent. of sterile cows in South Africa Statistics available from investigations carried out throughout the country show that endometritis chronica was found in 75 per cent. of the sterile females examined. The prevalence of this condition was further demonstrated in 68 cows destroyed because of incurable sterility. In 66 per cent. of these animals metritis chronica catarrhalis was demonstrable either macroscopically or histologically. Albrechtsen was the first investigator to point out the enormous rôle which this disease plays in sterility. His belief has since been supported by observers in most countries, Williams, Nielsen, Richter, Oppermann, Frei, and others. The condition is most frequently a sequel to puerperal metritis. It is, therefore, observed in cows which have calved or which have aborted. In South Africa it would appear to be uncommon in heifers which have not been served. One such case only has come to notice, that of an 18 months old heifer (Fig. 41, A.2) which showed cystic ovaritis and salpingitis, in addition to endometritis chronica. The heifer had never been served. Williams maintains that numerous heifers at breeding age are found to be sterile from endometritis chronica. He points out that this may be due to infection of the uterus during coitus prior to conception. Richter has not seen the disease in heifers which have not conceived. Oppermann has seldom seen the disease in virgin heifers. Kaufmann says endometritis chronica is commonly met with in chlorotic girls.

A history of affected animals usually reveals a puerperal metritis after the latest parturition, a retained afterbirth, or a difficult parturition which required assistance in delivery. In South Africa endometritis chronica is most commonly met with in herds where contagious abortion is widespread. Stoss states that the percentage of sterile cows in herds affected with abortion reaches over 20 per cent. In herds not affected with abortion the percentage is 10-12. Observations made in South Africa corroborate the statements of

Nielson, that sterility becomes most prevalent after abortion has been in existence in a herd for a few years and the cows have acquired some degree of immunity. The onset of abortion in a herd is ushered in with early abortions at the third or fourth month of gestation. At this period the afterbirth is not usually retained. The uterus cleans up quickly. The cow again shows oestrum and readily conceives six to twelve weeks post-partum. When the females in a herd acquire a degree of immunity to contagious abortion the foetus is carried seven or eight months or even to full time. The afterbirth is retained as a result of the incarcerating placentitis of abortion. Secondary infection gains entrance to the uterine mucosa through the portals kept open by puerperal metritis. Infection is also frequently conveyed to the uterus by laymen, whose handling of placental retention is for the most part irrational and always with insufficient antiseptic precautions. Another very important point in the causation of endometritis chronica is stable hygene. It is more widespread in byres where undesirable hygienic conditions prevail. It is a common occurrence to observe cows with puerperal affections after a normal parturition when housed under bad hygienic conditions. Sex hygiene also plays an important rôle. One frequently encounters in affected herds cows suffering from uterine affections, accompanied by uterine discharge, housed alongside apparently normal cows. This in itself would be sufficient to endanger the genital tract of healthy cows, but the danger is much exaggerated when such cows are served from time to time by the stud bull as they show oestrum.

Regarding abortion, it would appear to be evident that the bacillus of Bang is not of itself culpable as a causative agent in endometritis chronica. The statements of Richter, Nielson, and others, that it reduces the resistance of the uterine mucosa and opens the portals of the genital tract to secondary puerperal infections, would appear to be correct. Witt indicated the possibility that sterility may be caused by toxic products of Bang's bacillus developed in utero. It is well known with what rapidity the metritis of abortion in the absence of secondary infection disappears in cases of abortion which occur early during the gestation period. Further, these cases show oestrum at from six to nine weeks after abortion and as a rule readily conceive. The assertion of Nielson and Richter would, therefore, appear to be more likely than that of Witt.

The most common bacterial flora found in the non-gravid uterus, which shows macroscopic or microscopic pathological change, are *B. coli*, *B. pyogenes*, *Staphylococci*, *Streptococci*, *Micrococci*, *Streptococcus viridans*. It is easy to conceive, as Williams states, that any organism competent to cause wound infection may injuriously attack the uterus. A large percentage of cows which have been found to be suffering from chronic endometritis of old standing without marked uterine discharge have shown an entire absence of bacterial flora in the genital organs above the pars indivisa of the uterus.

Uterine catarrh is also observed accompanying new growths in the uterus. A few such cases have been observed. (Fig. 2, 55, 60, A. 2.)

It is not intended to discuss the symptoms of puerperal metritis; that is, metritis which occurs during the involutionary period of the uterus after parturition. It is, however, necessary to mention that the condition is exceedingly common in South Africa as a result of the causative conditions, already explained, existing in this country in a very exaggerated degree. A discussion of the symptoms of endometritis chronica as met with in sterile cows is considered sufficient.

During this discussion a sterile cow is looked upon as one which has failed to conceive after three or more services from a bull of known high fertility, or a cow at a clinical examination has shown uterine pathological changes known to be incompatible with fertility.

There are various degrees of intensity of endometritis chronica varying from a catarrhal to a purulent inflammation. The mildest form is a simple catarrh accompanied by little or no change in the size of the uterus and with no continuous discharge from the vulva. Such cases are quite impossible of diagnosis at a clinical examination as a rule. It is, however, possible to strengthen the idea that the condition exists for a few days after an oestral period when an opaque uterine discharge, which is not very profuse, will be evident. The cervix may be quite normal or the ostium uterinum may contain a small amount of mucus, which may also be found in small quantity in the cranial extremity of the vagina. A history of the previous parturition and puerperium will aid considerably in making a diagnosis.

Post-mortem examination of such a uterus shows little macroscopic change in the mucosa. The change is frequently confined to one horn, which is usually somewhat larger than the other, or it may be bilateral. In cases of such a chronic nature the mucosa is smooth, moist, slightly transparent, greyish-pink to greyish-blue in colour. It appears to be somewhat thickened and oedematous. In old cases the affected mucosa is smooth and shows traces only of the normal cotyledons of the non-pregnant uterus. There may or may not be a small accumulation of opaque tenacious mucous exudate in one or both horns. In many of these cases, there is no apparent macroscopic change. Histological examination, however, shows that chronic inflammation exists.

The next stage in intensity is somewhat less common than that which has been described. There is a more or less continuous discharge from the genital tract which soils the tail and buttocks. It is especially marked for three or four days after an oestral period. Vaginal examination shows the presence of an opaque or clear tenacious mucus with some yellowish or yellowish white flocculi accumulated in the cranial extremity of the vagina. The cervix may or may not be the seat of pathological change. The ostium uterinum contains some tenacious mucus mixed with flocculi. The uterus and uterine horns are somewhat enlarged, with flaccidity of the wall, which may be somewhat thickened. There is a loss of tone and slow reaction to palpation. Affected cows frequently show irregular oestral periods, with abnormally long interovulation periods, or again the oestral periods may be at normal intervals of 20 to 22 days. It is not uncommon to find tubal and ovarian affections concurrent in cases of endometritis chronica of this nature.

Post-mortem examination shows a well-marked macroscopic change in the uterus. On section there is a small accumulation of tenacious mucus with purulent flocculi in the horns. The pathological change may be confined to one or both horns. The uterus is somewhat enlarged. One horn is frequently larger than the other. The mucosa is greyish-pink, greyish-blue, or greyish-brown in colour. It is smooth, moist, somewhat swollen, and semi-transparent. There is evidence of oedema. In old-standing cases the normal cotyledons of the non-pregnant uterus may have undergone degeneration, traces only being evident as greyish-yellow areas through the mucosa, or complete degeneration may have taken place; the situation of the cotyledon being represented by an oval depression in the mucosa varying in size from 2 to 4 mm. in length, 1 to 2 mm. in width, and ½ to 1 mm. in depth. Associated with the changes in the uterus it is not infrequent to find hydrosalpinx, cystic pavilionitis, and cystic ovaritis. The lesions in the salpinx are often not apparent without a histological examination.

Cystic degeneration of the ovaries, as already mentioned, is often found associated with endometritis chronica. Hess maintained that the pathological changes found in the uterus of nymphomaniac cows were probably secondary to cystic degeneration of the ovaries. There appears to be little doubt but that the pathological change in the ovaries is a sequel to endometritis as a result of extension of infection from the uterus through the fallopian tube to the ovary. It is easy to understand how infection of the ovary can be accomplished through this route, especially through the crater of a ruptured Graafian follicle. The probability of ovarian infection being culpable for cystic degeneration of the Graafian follicle has been pointed out by Williams, Richter, Wester, and others. It is unlikely that there is a predisposition to cystic degeneration of the ovary in cows of different breeds, neither is it likely that climate, environment, or feeding plays a direct part. Cystic degeneration of the follicle is frequently associated with nymphomania. Observations in this country convince one that all ovaries which show cystic degeneration of the follicle or cysts which cannot be differentiated either macroscopically or histologically from Graafian follicle cysts are not associated with nymphomania. In fact, a fair percentage of cows showing these cysts in the ovary show no nymphomaniac symptoms whatever. Further, nymphomania has been seen, certainly without marked change in the pelvis, in animals sterile from other causes than cystic ovary. A number of the cows affected with cystic degeneration of the Graafian follicle showed "anaphrodisia."

The condition nymphomania is quite characteristic and well known to all veterinarians who interest themselves in the diseases of the genitalia of cattle. (Figs. 62, 63, A.2.) The symptoms of nymphomania do not as a rule develop suddenly, although a sudden onset has been observed. It appears gradually; the interovulation periods becoming shorter and shorter and each oestral period longer in duration. The pelvis becomes changed in appearance. The sacrosciatic ligaments become relaxed and sunken. The root of the tail becomes markedly elevated and coarse looking, leaving a marked depression on either side. The tail as a rule hangs to one side. The back becomes hollowed at the lumbo-sacral articulation, while the dorsal spines present a convex line. The head and neck become coarse and ox-like. The voice becomes deep, resembling that of a bull. The external genitals become enlarged and flabby. The milk yield becomes decreased, and its quality also deteriorates. The animal's temperament becomes changed. There is unusual sexual desire. The cow pays abnormal attention to other cows in oestrum. She will show abnormally short interovulation periods, or she may allow copulation to take place at any time. Clinical and post-mortem examination show that oestrum is not necessarily associated with ovulation. Wester states that the Graafian follicles ripen quickly and can become fully developed in a week after the last ovulation. He further indicates that ovulation may occur without oestrum, and oestrum without ovulation. When ovulation does occur the resulting corpus luteum develops rapidly, is larger than normal, soft and oedematous, and is easily expressed according to Wester. Our observations support those of Wester. The cow loses condition. This train of symptoms may continue for a considerable period. The final result is seldom observed, as the cow is sold as incurably sterile or is castrated. Nymphomaniac cows have been kept under observation at this Institute for a considerable period. It appears that as the cysts increase in size at the expense of the ovary tissue, which undergoes pressure atrophy, the symptoms of continuous or frequently recurrent oestrum disappear, often altogether for several weeks or months, but the tendency to mount other cows remains.

The clinical examination of the genitalia of the nymphomaniac bovine is characteristic. The vulva is enlarged, flabby, and admits the hand without difficulty. The vagina usually contains a quantity of tenacious opaque mucus. The first cervical fold is frequently ectropic, swollen, and ragged. The second cervical fold may also be visible through the ostium uterinum externum. The ostium uterinum is usually open and admits the finger to its entire depth. It contains a characteristic opaque tenacious mucus. The uterus is enlarged and its wall flabby. It is lacking in tone, and responds slowly or not at all by contraction to palpation. The horns are also flabby and hang well forward in the abdomen as a result of stretching of the broad ligaments. One or both ovaries are enlarged and cystic. Hydrosalpinx is a not uncommon concurrence. Our experience proves that the cystic degeneration of the ovary associated with nymphomania is concurrent with macroscopic or microscopic change in the tube.

The post-mortem appearance of the uterus is similar to that described as endometritis chronica catarrhalis and endometritis chronica muco-purulenta.

There is another chronic pathological change in the uterus which has been observed in several cases of nymphomania of old standing, that is endometritis catarrhalis chronica cystica. (Fig. 17, A.2.) This cystic change in the uterine mucosa has been met with most frequently associated with nymphomania, but it has also been met with in incurably sterile cows, associated with salpingitis and cystic ovary, which did not show nymphomania. Cystic endometritis has been met with only in cases of old-standing sterility of endometritic origin, and in one case of hydrometra. In the early stages of cystic degeneration it is not possible to make a definite diagnosis as to the presence of the condition at a clinical examination. There is an accumulaton of some opaque mucus with some flocculi in the vagina. The cervix is enlarged and as a rule somewhat sclerotic. The mucosa of the cervix may show small cysts. On rectal palpation the uterus and uterine horns are enlarged, soft, and markedly lacking in tone. There is slight or no reaction to uterine palpation. In the early stages when the cysts are small it is impossible to feel them in the uterine wall, but large cysts which have sometimes been met with (Fig. 17, A.2) can be easily palpated. As already mentioned, hydrosalpinx and cystic ovaritis are frequently concurrent. It has been associated with all our cases of cystic degeneration of the uterine wall.

Post-mortem examination shows the uterus to be enlarged with flabby walls. In the majority of cases it is possible to feel the cysts in the uterine wall; sometimes large cysts develop which are easily palpable. These large cysts may pass through the entire wall so that they become visible on the serous surface as semi-transparent rounded elevations with a thin capsule. (Fig. 17, A.2.) On section the mucosa presents a moist greyish-yellow to greyish-brown colour. It is swollen and oedematous. The horns of the uterus contain a small quantity of opaque mucus. There is no evidence of the cotyledons of the non-pregnant uterus. They have undergone complete degeneration. The mucosa presents cystic degeneration. The number and the size of the cysts vary considerably. There may be only a few cysts (Fig. 3, A.2) or the mucosa may be more or less closely studded. (Fig. 17, A.2.) The cysts vary in size from a pin's head to a pea, but isolated large cysts have been seen measuring up to 4 cm. in diameter. These large cysts frequently penetrate from the mucous to the serous surface. The cysts appearing on the mucous surface are greyish semi-transparent rounded elevations with a thin transparent capsule. On section of the uterine wall when the cysts are numerous, there is a honeycombed appearance. (Fig. 17c, A.2.) The contents of the cysts is liquid, greyish, or semi-transparent in colour. They are almost entirely confined to the mucosa, but the larger cysts penetrate through to the serosa or into the muscular coat. This form of degeneration is for the most part confined to the horns of the uterus, but isolated cysts have been seen in the mucosa of the pars indivisa.

In those cases which have been observed here the cervix has been the site of sclerotic change with some stenosis of the lumen towards the cranial fold. The mucosa has also shown the presence of some small cysts. (Fig. 17c, A.2.)

This form of cystic degeneration would appear to be due to occlusion of the mucous glands of the uterus as a result of endometritis. Halter has pointed out that cysts have also been seen on the cotyledons. He therefore maintains that all uterine cysts are not developed from the uterine glands. This condition would appear to be somewhat similar to that described by Steck as endometritis cystica heterotopica.

There is a further form of endometritis chronica in which more marked pathological change is evident, namely, endometritis chronica purulenta or pyometra. In this case there is an accumulation of pus in the uterus either as a result of stenosis or atresia of the cervix or complete lack of tone in the uterine wall. It results from lesions already described under the general causes of metritis. Foetal maceration has been indicated as a frequent cause (Krupski). The condition would not appear to be common in this country. Four such cases only have come to notice. This represents less than 1 per cent. of the cattle examined for sterility. Richter has met the condition in 0.5 per cent. of his cases only. Albrechtsen and Williams, however, have frequently seen the disease in their enormous material. Hess believed that contagious granular vaginitis resulted in some cases in a pyometritis. This is also the belief of at least one observed in this country (Jarvis). It appears, however, that the more likely origin of the disease was not excluded. Richter states that contagious granular vaginitis as a cause of pyometritis is extremely unlikely. This is the belief of all well-known authorities throughout the world at the present time. Observations carried out on hundreds of cases of contagious granular vaginitis in this country indicate that purulent inflammation of the uterus cannot be associated with the former condition. Careful investigations into the history of such animals always prove the presence of puerperal affection after the last parturition. In one case maceration of the foetus had occurred about the fifth month of gestation.

The symptoms associated with pyometra are very evident. When there is much pus accumulated in the uterus, a certain amount of general disturbance results from toxic absorption. The animal remains unthrifty although it feeds well. (Fig. 28A, A.2.) When little pus accumulation occurs, there is as a rule no general disturbance. There is no appearance of oestrum. Richter states that oestrum may infrequently appear. However, in the four cases which have been observed here there were no oestral periods. The ovaries when examined post-mortem showed embedded retained corpus luteum in one ovary and no evidence of Graafian follicles near maturation in the other ovary, so that ovulation was unlikely. When the cervix remains open there is a constant or periodic discharge from the genital tract which soils the tail and buttocks. This discharge is most marked when the cow lies down; a quantity of purulent material accumulates on the floor of the byre. The discharge is thin or thick-liquid yellowish pus, which is as a rule not offensive; occasionally it may be very offensive. It is sometimes passed during periods of straining.

On rectal examination the clinical findings vary considerably. When the cervix is open so that the purulent exudate can escape, the uterus is found enlarged, especially the horns, which are as a rule irregular in size. One horn may be larger than the other. The wall is thick and firm. There may be peritoneal adhesions between the uterus and the broad ligaments or between the uterine horns and the ovaries or the mesosalpinx.

When the uterus has lost tone or when the ostium uterinum is stenosed or occluded so that the free exit of pus is not possible, the exudate accumulates in the uterus. It varies considerably in amount up to several litres. From one cow 21 litres of pus were removed. (Fig. 1, A.2.) The uterus hangs over the pelvic inlet. Its size depends upon the accumulated pus. It is enlarged and thin-walled. The horns are as a rule uniformly enlarged and their extremities are rounded. It is possible to demonstrate fluctuation. When complete occlusion of the ostium uterinum has occurred, the uterus may be enormously enlarged, thin-walled, and very tense.

The cervix is always the seat of pathological change, marked cervicitis, hypertrophy, sclerosis, or complete occlusion may be met with. If it be possible to palpate the ovaries, a corpus luteum will as a rule be present, but it may be difficult to palpate as it is frequently embedded (Williams). Williams's statements have been supported by the observations at this Institution.

The possibility of mistaking the pyometric uterus for a pregnant uterus has been pointed out by Williams, Richter, and others, but careful examination eliminates the possibility. There is an absence of the normal uterine plug of pregnancy. Cotyledons cannot be felt. Both horns are as a rule equal in size. The pulsation of the uterine arteries is not characteristic of pregnancy. However, it is readily admitted that mistakes in diagnosis may easily occur in a cursory clinical examination.

Sclerosis of the uterine wall as described by Williams has been met with. (Fig. 18A, A2.) The case was a young cow which had suffered from pyometritis for six months before she was submitted for examination. Her cervix was difficult to penetrate, as it was the seat of an acute cervicitis. The uterus contained 2 litres of foetid pus. The cow was treated several times over a period of two months. The pus accumulation ceased, but the uterus gradually became hard, almost cartilage-like, especially the horns, which in parts were 3 cm. thick. The whole wall was markedly but irregularly thickened. The mucosa of the uterus and the cervix was the seat of large ulcerated patches. There was no evidence of cotyledons.

The wall on section was yellowish-white in colour and extremely firm. It was thickened throughout, but the thickening was irregular in some places. The walls varied from 1.5 cm. up to 3 cm. diameter

The pyometric uterus on post-mortem examination is found enlarged. When little pus accumulation is evident, the wall is thickened and firm, especially that of the horns, the apices of which are usually rounded. When there is much accumulation of purulent exudate, the wall is thin and the uterus fluctuating. The horns are frequently symmetrical, but are also found unequal in size. Frequently adhesions are present on the serous surface of the uterus, and horns, through which it is adherent to the broad ligaments and mesosalpinx, or fibrous tissue adhesions may exist between the ovaries and the uterine horns. On section the organ contains more or less accumulation of pus varying in amount up to several litres. The pus may be thick or thin liquid, usually non-foetid, but it may also be very foetid. It is yellowish or yellowish-grey in colour. The mucosa is greyish-brown or greyish-red in colour, showing adherent purulent flocculi. It may show ulcerated patches over which the epithelium has been removed. The cotyledons are swollen, reddish-brown or greyish-brown in colour. They may be smooth on the surface or roughened with epithelial defects. In old-standing cases the cotyledons may have entirely disappeared, and the mucosa presents a nodular appearance, the nodules being placed close together. (Fig. 35, A.2.) In some cases, where the purulent exudate has not entirely filled the organ, a marked line of demarcation is evident at the upper limit of the contents. The mucosa towards the cervix being smooth and pink. (Fig. 35, A.2.)

Histology of Endometritis Chronica Catarrhalis.

In the case of endometritis chronica catarrhalis the epithelium is intact or may show epithelial desquamation. There may be attempts at regeneration as shown by the local formation of a stratified epithelium, several cells thick. The mucosa is thickened, frequently the seat of marked oedema (Fig. 18, A.3); the interstices of the connective tissue stroma being filled with fluid. There is infiltration with lymphocytes and eosinophilia in the epithelium and in the mucosa propria. The stratum cellulare is less rich in cells than normal. The connective tissue fibres in the stroma are increased in number. In many of the sections examined a large cell with small acidophyle granules and a round or slightly oval nucleus was observed in the mucosa. The epithelium of the mucous glands may show desquamation and there may be accumulation of cell débris in the lumen. (Fig. 3, 6, A.3.) Reduction in the number of the glands from pressure of the increased connective tissue stroma is usually evident (Wester). When there is marked oedema, the glands sometimes show group isolation (Fig. 18, A.3), the groups being surrounded by loose connective tissue strands bathed with fluid. The lumen of the glands is sometimes enlarged, apparently the result of retention of mucous secretion from blocking of the gland-opening. It is not unusual to find thickening of the walls of the capillaries in the mucosa and hyaline degeneration. In one case there was marked arterio-sclerosis of some of the vessels. (Fig. 26, 27, A.3.) Such change in the vessels has already been described by Kaltenegger and Wester. The zona muscularis is as a rule not changed.

Histology of Endometritis Chronica Catarrhalis Cystica.

The epithelium shows evidence of regeneration. It may show a normal single layer of cylindrical cells without cilia, or here and there may be evidence of stratification. Occasionally there are local areas of epithelial desquamation. The mucosa propria may show round cell infiltration and increase in connective tissue. The propria is less rich in cells than the normal propria mucosa. The glands may in part show a normal epithelium, or there may be others showing epithelial desquamation with cell débris in the lumen. (Fig. 4, A.3.) In the neighbourhood of the cysts the glands are frequently decreased in number and deformed by their walls being pressed together. The cysts are for the most part confined towards the epithelial lining of the mucosa, the majority of glands being situated towards the zona muscularis. Cysts, however, are also found in the deeper parts of the propria. (Fig. 12, A.3.) The cysts vary in size and shape, some are rounded, some oval, and others irregular in shape. (Figs. 15, 16, 17, A.3.) They are lined with a single layer of cubical to flattened cells. The larger cysts almost always have a flattened epithelium. It is not unusual to find the walls of the capillaries in the mucosa thickened and occasional vessels may show hyaline degeneration. The cysts are for the most part confined to the mucosa, but some large cysts pass through the muscularis and reach the serosa. (Fig. 17A, A.2.) These large cysts show a markedly flattened epithelium.

The zona muscularis is unchanged except in the neighbourhood of the large cysts when there is a local muscular atrophy. (Figs. 12, 13, A.3.)

The condition appears to be that of retention cysts due to blocking of openings of the uterine glands during the acute metritis, which is nearly always the origin of endometritis chronica. The cysts are at first confined to the mucosa, but later, as the result of pressure and consequent atrophy of the zona muscularis, many of them reach the serosa.

Histology of Endometritis Chronica Purulenta: Pyometra.

The epithelial layer of the mucosa is entirely or partly absent, resulting in superficial necrosis or ulceration. Occasionally attempts at regeneration are evident. The epithelial layer shows local thickening. It may be several cells thick. The mucosa is covered with a purulent deposit containing neutrophiles and cell débris. It is infiltrated with neutrophiles and round cells. There is increase in connective-tissue stroma. Towards the surface there is a decrease in the number of mucous glands, or glands may be entirely absent. (Figs. 23, 24, A.3.) Groups of mucous glands are seen towards the muscular coat in the mucosa propria. Some of them are irregular or flattened in shape. They are surrounded by connective tissue which is infiltrated with neutrophiles and round cells. (Figs. 20, 22, A.3.) The gland epithelium in part shows desquamation and the lumen is filled with débris in which can be recognized neutrophiles and round cells. The calcified areas described by Millo have not been seen. Thickening of the capillary walls is frequently met with.

When enormous distension has taken place, the muscular coat is thin as a result of atrophy.

The zona muscularis may also be the site of inflammatory change (Richter).

In one case a mixed endometritis chronica catarrhalis cystica and endometritis chronica purulenta was met with. The mucous glands in part were typical of what has been described in the former condition. Some neighbouring glands, however, were very much dilated. The epithelium had for the most part disappeared or showed marked desquamation. The lumen was filled with neutrophiles and cell débris. In most glands the luminal contents had undergone inspissation and disintegration in the centre, and presented a homogeneous mass, which showed areas of calcification. This was surrounded by closely packed neutrophiles. (Fig. 25, A.3)

Sequelae to Endometritis Chronica.

Endometritis chronica either in the catarrhal or purulent form is a very important disease from the point of view of the breeder. Cows suffering from the condition are sterile either as a result of failure to conceive or as a result of early abortion, due to failure of the embryo to become attached to the uterus, because of irreparable pathological change. It is not difficult to conceive that the exudate from the pathological mucosa of the metritic uterus is destructive to spermatozoa. The researches of Wester indicate that the production of spermatoxin is highly probable. In cases where metritis has existed so long that the cotyledons have entirely disappeared from the mucosa of the uterus, the physiological importance of these areas in the implantation of the embryonic envelopes is lost. The embryo remains unnourished and is expelled early.

It is quite possible that there is a tendency to spontaneous recovery, and that such does occur is without doubt. However, in many cases spontaneous recovery does not take place, and unless the condition is treated early incurable sterility is the result.

Chronic tubercular metritis is incurable. As pointed out by Richter, it is probably far more common than it is taken to be. In 49 cases of chronic endometritis mentioned by Richter, six were proved to be of tubercular origin.

Pyometra is always very serious. Cases of long duration are incurable. The less acute uterine catarrh reacts fairly readily to suitable treatment provided active steps are undertaken before irreparable pathological change has taken place in the mucosa. Richter states that as a rule over 50 per cent. of cases of endometritis chronica catarrhalis can be cured and the animals again become pregnant. During these investigations the results obtained in the treatment of chronic uterine affections by the methods described indicate that the percentage of cures given by Richter is a somewhat conservative one. Treatment should be begun as early as possible if the best results are to be obtained.

Prophylaxis is of the utmost importance in addition to direct local treatment of the genital tract.

When the causes of endometritis are considered it will be seen what an important rôle hygiene plays in the production of endometritic sterility. Therefore, the first consideration should be maintenance of ideal hygienic conditions in the byre. The most ideal hygienic arrangements should also be observed in milking. Sufficient exercise at pasture is also an excellent way of attaining the highest degree of bovine vitality and consequent resistance. When the weather is favourable, cows should be allowed to calve in the open, provided a grass paddock is available. Under South African conditions, where " the open " means intense heat and dust or great variations in temperature, it is preferable that a special large roomy calving box should be available. The calving box is a blessing when kept properly clean, but it becomes the reverse in the absence of good hygiene. It should be used only as a calving box and should be disinfected after each calving. It should be bedded with a liberal supply of clean dry straw or veld hay. Cows after calving should not be returned to the herd for a couple of weeks. They should be kept isolated in clean quarters or run during the day in a small grassy paddock. The puerperal secretions which soil the tail and quarters should be washed off daily. Cows which retain the foetal membranes, always an indication of metritis, should be treated in the most up-to-date way for this condition. Cows must not be allowed back into the stable until all discharge from the genital tract has ceased. Animals suffering from suppurating conditions, such as panaritium, should be removed from contact with healthy cows. Under no circumstances should a cow showing a discharge from the genital tract be served.

It is essential that the beginning of all treatment for sterility should be the maintenance of cows on a properly balanced ration, known to be compatible with high fertility. This precaution is especially to be observed in the beef breeds and in dry cows of the milking breeds where there is a tendency to put on too much fat. Fresh green food is an essential element in the ration fed to breeding cows if the maximum percentage of fertility is to be maintained. Recent work of Graves and Miller has shown the value of vitamin E in cases of functional sterility.

Since contagious abortion would seem to be the most fertile cause of general sterility in bovines, the herd should be tested serologically to ascertain the extent of infection. If infection is more or less general, all non-pregnant females should be vaccinated about three months before it is intended that they should be served. If infection is found to be limited to a small percentage of the herd, it is a better policy to try and eradicate the disease by serological tests carried out at intervals of four to six weeks.

Regarding direct uterine therapy, it is without doubt a somewhat tedious procedure when endometritic sterility is widespread, but it is absolutely essential to success. Ovarian treatment alone has not met with success in South Africa. All instruments used in the treatment of sterility should be sterilized prior to use.

The cervix is withdrawn to the vulvar opening with a cervical forceps. The forceps used is that recommended by Williams, but there are many suitable retraction forceps obtainable. The next procedure is to ascertain the patency of the cervical canal with a small dilator. If it is normally patent, it will admit a small irrigation catheter. When the canal is abnormally small, it can be dilated with a dilator so as to admit the catheter. There are cows in which it has been found impossible to pass the dilator through the cervix. In these cases Williams' recommendation has been adopted with success, that is, to await an oestral period. When passage through the canal is not possible with the instruments available, it is recommended that the opening should be enlarged with the knife. This operation has never been found necessary during these investigations. Richter recommends that "the cause of the stenosis should be removed with the knife or scissors, and prominent folds incised or cut away." The passage of the catheter must be undertaken with the utmost care and the sinuous canal followed without pressure, as the mucosa or the wall of the cervix may be easily perforated. For success in the use of the uterine catheter an intimate knowledge of the anatomy of the cervix is necessary. Anatomists and obstetricians, including Williams and Richter, have described the cervical canal as not straight, but inclined to be spiral or S-shaped with 3 or 4 circular folds of mucosa. These folds sometimes form a spiral twist like the thread of a nut. Williams and Richter point out that in the majority of cases the direction of the spiral is downward and towards the right from the ostium uterinum externum. It is more easily attained when the cervix is withdrawn and grasped in the left hand which has been placed into the vagina. This is, however, unnecessary in a cervix of average patency. There are several suitable catheters obtainable. The most suitable appears to be that recommended by Richter and Nielsen, both of which have an introduction and a withdrawal tube.

By controlling the passage of the catheter through the uterus by the left hand in the rectum, the apex being directed towards the lateral wall, it is introduced deeply into the uterine horn. Oppermann considers it unnecessary to introduce the catheter deeper into the uterus than the pars indivisa. It is safer, however, to introduce it deeply. The irrigation fluid is conveyed into the uterus by means of a rubber tube and a funnel which is attached to the uterine catheter. Small quantities, 100-150 c.c., of fluid are introduced and the horn then massaged. This is done three or four times and the fluid withdrawn. The other horn is irrigated in a similar manner, the catheter having been withdrawn to the pars indivisa of the uterus and then introduced into the second horn.

As an irrigant Richter and Albrechtsen recommend normal saline. Williams recommends ½ to 1 per cent. Lugol's iodine solution in normal saline. Nielsen has used Lugol's iodine and rivanol solutions with good results. Lugol's iodine solution 1 per cent. in normal saline has been used in South Africa with success. The irrigation has been followed by a few c.c.'s of pure Lugol's iodine into the apex of each horn, as recommended by Richter. After removal of the catheter, the uterus is gently massaged. The operation is completed by swabbing the whole cervical mucosa with pure Lugol's iodine, as recommended by Williams. Nielsen has treated endometritis catarrhalis with success by injection, into the uterus, of solutions of iodine and rivanol, varying in strength from one to ten per mille. The quantity used depended on the capacity of the uterus, up to 200 c.c.

After irrigation has been completed, it is advised that a disinfectant and astringent be introduced into each horn. Albrechtsen used kreolin, lysol, lysoform, and alum in various strength solutions. He pointed out that strong solutions are contra-indicated and have no more influence on the condition than weak solutions. Strong disinfectants cause marked straining, which may continue for hours. Other disinfectant and astringents which have been used with success by various workers are chinosol, 1 per cent.; tincture of iodine, 2 per cent. in water; pyoctanin, 1 per cent.; kolapo, 1 per cent.; and yatren, 1 per cent. solution.

During treatment of the uterus the vagina should be irrigated with normal saline three times a week to remove any exudate which may accumulate at its cranial extremity.

In the treatment of pyometritis the uterine contents should first be drained off. It may be necessary to dilute it with saline solution as recommended by Williams. After the purulent contents have been removed, the uterus is irrigated several times with 1 per cent. Lugol's iodine in normal saline or 1 per cent. rivanol solution.

After irrigation, Williams recommends introducing 8 to 16 ounces of liquid paraffin, holding in suspension half an ounce each of iodoform and bismuth. This treatment has been tried in this country with success.

In the case of light catarrhal endometritis, three or four irrigations at intervals of a week or two weeks are usually sufficient in those cases which react to treatment. There is a small percentage of cows which suffer from catarrhal metritis which do not react to treatment but continue sterile. In pyometritis several irrigations are necessary. It may be necessary to continue treatment over several weeks.

Cysts when present in the ovaries should be ruptured by vaginal pressure. The corpus luteum present in most cases of pyometra should be expressed if possible at the first treatment.

It appears to be a good general rule to massage the uterus and ovaries during the course of treatment and for a couple of weeks afterwards.

The experience in this country supports the statements of Williams Albrechtsen, Richter, and others that ovarian treatment alone is not of itself sufficient to bring about cure in cases of endometritis chronica. There are cows which do recover with ovarian treatment alone—that is, rupture of ovarian cysts and expression of retained or cystic corpora lutea, but the percentage of such recoveries is relatively small when compared with those treated by the combined method of direct uterine and ovarian treatment.

Oppermann recommends in certain cases of endometritis chronica, which do not react to the methods of treatment just described, the use of specific-unspecific protein therapy. Under this instruction a considerable amount of work has been carried out by his students in the Tierärztliche Hochschule, Hanover, on this method of treatment. He recommends yatren vaccine. The Behring-Werke in Marburg put on the market a special yatren vaccine suitable for cattle. It is apparently a polyvalent vaccine, containing organisms most commonly found in the flora of the pathological genital tract. The vaccine is given subcutaneously in doses up to 50 c.c. It causes a local reaction on the genital tract in the way of increased secretion from the mucosa, which passes off during the course of the treatment. Oppermann points out that this treatment apparently produces a tendency to resolution. He, however, indicates that the prognosis is naturally better when treatment can be begun early, and that the hope of success is not great when old cases of pyometra are receiving attention.

In 1920 Albrechtsen tried electrolysis in the treatment of endometritis chronica. Albrechtsen claimed excellent results, but stated that it was a difficult and tedious procedure. Zeeh, working under Richter's instructions, tried the use of electrolysis in 1922. He treated twenty-three sterile cows with apparently excellent results, fifteen cows recovered after one treatment; two cows recovered after two treatments; two cows had to be slaughtered; three with cystic degeneration of the ovaries concurrently with endometritis chronica were not influenced by treatment; one cow suffering from acute endometritis was also uninfluenced in spite of being treated three times.

The uterus is filled with 1 per mille. copper sulphate solution by means of a catheter. The anode is introduced into the uterus and the cathode connected. The strength of the current could be increased at will; treatment was continued for five minutes in each horn. Fine particles of copper were deposited on the uterine mucosa, due to breaking down of the copper sulphate in the solution. The treatment produces contraction of the uterine wall and causes the animal uneasiness for some time. Richter agrees that the method is difficult and tedious.

(3) Hydrometra with Cystic Degeneration of the Uterine Wall.

Hydrometra (with cystic degeneration of the uterine wall) appears to be a very rare condition in bovines. It has been met with in one case only. (Fig. 11, A.2.) The subject was a cow which had never conceived owing to congenital deformity of the cervix. It is difficult to say whether the ostium uterinum was ever really permeable for the passage of the oestral débris. With such a deformity it would have been extremely difficult. At the time of examination the cervix was sealed with a false uterine seal which caused complete closure. The cause of hydrometra would appear to be cervical deformity, which rendered the escape of oestral débris through the ostium uterinum impossible. Williams mentions having met with a similar case, but the cervical canal was open and admitted the uterine catheter. This was followed by cystic degeneration of the mucous glands. Vaginal examination may reveal a false uterine plug or a cervical deformity. Such a deformity may be either congenital or it may be the result of parturition injuries. On rectal examination the uterus is entirely abdominal. It is enlarged and fluctuating. The wall is thin and lacking in tone. Both horns are equal in size. Palpation does not indicate the presence of an embryo. The pulsation of the uterine arteries is not palpably increased in volume, nor are the arteries increased in thickness. There is no trace of cotyledons to be felt. The fallopian tubes are not changed. The corpus luteum of the last interovulation period will as a rule be present, but it may not be palpable through the rectum. The broad ligaments are stretched. The case observed at this Institute showed an oestral period, with a copious discharge of opaque viscid mucus, once during 12 months; the oestral period was accompanied by the rupture of a Graafian follicle. The fluid did not accumulate in the uterus very rapidly after its partial expulsion. It was three months before the accumulation was again so extensive as formerly.

On post-mortem examination the uterine wall is thin and flabby. The cavum uteri is increased in size according to the extent of the accumulated fluid. The contents are thin, viscid, opaque, non-foetid, mucous material. The mucosa is pale pink, smooth on the surface and moist, or it may present a wrinkled appearance. (Fig. 11, A.2.) The cotyledons of the normal non-pregnant uterus are not present. There are occasional small transparent cysts scattered over the mucosa. (Fig. 11, A.2.) They project somewhat above the surface of the surrounding mucosa, they are thin-walled and confined to the mucous coat. They do not appear to penetrate the muscularis. The cysts were not nearly so numerous as in the case described by Williams.

Microscopically the epithelium is high columnar and the cells are heavily charged with mucus. The mucosa is very narrow. It appears to be less than half the thickness of the normal uterine mucosa. There is some increase in fibrous tissue; this is especially marked towards the zona muscularis. The uterine glands are very much decrease in number; some are enlarged, showing the commencement of cystic change. The glandular epithelium is high cubical. The lumen appears filled with mucus.

The largest cysts are lined with a cubical or flattened epithelium. They project above the surrounding epithelium and here and there can be seen to penetrate a short distance into the zona muscularis. In a few places in the section the fundus of the glands could be seen entering the muscular layer, and at places where the glands were cut across they appeared as isolated glands in the zona muscularis. (Fig. 8, 9, A. 3.)

The prognosis in such a case would be hopeless. When a diagnosis of hydometra of this nature has been established the animal should be prepared for slaughter.

(4) TUBERCULAR METRITIS.

Tuberculosis of the uterus is a common concurrence with tubercular lesions in the peritoneum and lungs in bovines. It has been found in 18 per cent. of tubercular cows destroyed during these investigations. The percentage of tubercular cows affected with genital lesions recorded by different observers differs greatly. Lungwitz found 58 per cent of cows suffering from peritoneal lesions with concurrent uterine tuberculosis. Ostertag found 65 per cent. of cows with generalized tuberculosis suffering from genital lesions. Meyfarth recorded 11.04 per cent. Büchli recorded 17 per cent. Winkel recorded 22.5 percent. out of 7,517 cows slaughtered because of open lesions in the lungs. Uterine tuberculosis in the absence of open tubercular lung lesions was recorded by Winkel in 35 cases only out of the enormous material slaughtered by him in the Netherlands during the years 1905-7. Herschel, working in the Berlin slaughter-house, found 6.47 per cent. of cows with general tuberculosis suffering from tubercular metritis. Krupski recorded 24 per cent. of tubercular cows as showing uterine lesions.

Pathogenesis.

The most common route through which the bacillus reaches the uterus appears to be the fallopian tubes, from an infected peritoneum. This route of infection is agreed upon by most observers, including Frei, Aschoff, and Kaufmann.

There is no doubt that infection also occurs through the blood-stream. In this case, however, one must expect a miliary tuberculosis of the uterus (Aschoff). It is probable that the bacilli reach the uterus through the blood-stream in generalized tuberculosis. The bacillus frequently reaches the uterus through other routes as well as the fallopian tubes. This is supported by the fact that tubercular lesions in the uterus are occasionally met with in the absence of tubal tuberculosis.

Infection can also occur as an extension through the muscular wall from the uterine peritoneum per continuitatem. This is probably a common route when tubercular perimetritis coexists with peritoneal tuberculosis. There would also appear to be tubercular infection of a primary nature by extension from the external genitalia. However, it would appear that primary tubercular metritis is extremely rare (Kaufmann, Aschoff). The majority of cases are secondary in nature. Frei states that the secondary nature of the uterine lesions can as a rule be established by the fact that the lesions are older and more pronounced towards the ovarial extremity of the tubes. Some of the cases which have been examined here have shown younger lesions in the uterus, even in the absence of tubal lesions, than the lesions in other organs. However, in the majority of cases Frei's views have been supported. The lesions decreased in number and age from the apex of the horn to the cervix. During these investigations tubercular metritis has not been met with in the absence of tubercular lesions elsewhere.

Primary infection in this country would appear to be of rare occurence. That such a possibility of transmission by coitus exists must not be lost sight of, but tubercular lesions in the genitalia of the bull have only once been encountered at this Institute. Frei and Williams draw attention to the possibility of transmission during the manipulation of the female genitalia in parturition or in the treatment of sterility. That there is a probability of transmission during the treatment of sterility cannot be doubted, especially when antiseptic precautions are not all that can be desired. In the early stages of tubercular invasion of the uterus diagnosis is not easy and it may be mistaken for a metritis chronica catarrhalis and treated as such. Infection with the catheter during uterine irrigation possibly takes place frequently.

Symptoms.

The insidious nature of tubercular invasion does not allow of an early diagnosis. The disease must be well established before it can be detected. The first symptom is the appearance of a slimy catarrhal discharge from the vulva, which is continuous. The discharge is opaque, catarrhal to muco-purulent in nature, but not foetid. It gradually increases in volume and may show blood-streaks. The tail and buttocks are continually soiled. It appears to increase during and for four to five days after each oestral period. Williams says that the discharge is greatly increased if copulation is allowed. Owing to the frequency with which the tubes and the ovaries are involved the oestral periods are usually irregular with abnormally long interovulation periods. Sterility and nymphomania, with its resulting deformity of the rump, is also a common sequel. Williams mentions that the uterine discharge is favourably influenced by uterine irrigation, but the improvement is not long maintained.

Rectal examination allows of a more intimate knowledge of the pathological changes, and with the aid of the tuberculin test usually permits a correct diagnosis. At the onset rectal palpation does not reveal any change in the uterus. It is not increased in volume. However, since it is the rule that the tube is first involved, tubal palpation may establish the tubal lesions. The somewhat characteristic tubal lesion, the uterine catarrh, the tuberculin test, and microscopic examination for tubercle bacilli, usually enable a definite diagnosis of uterine tuberculosis to be established in an early stage of the disease. As the pathological changes become more pronounced, the uterine horns increase in size and the wall becomes firmer. Careful palpation reveals the presence of tubercular nodules in the wall. As a rule the nodules can first be felt towards the apex of the horn. It is not uncommon for one uterine horn to become larger than the other. However, both horns are as a rule symmetrically involved. At this stage the discharge is profuse, muco-purulent, and non-foetid. The neighbouring sacral lymphatic glands are frequently involved. They are firm and enlarged.

The ducts of Gaertner are sometimes invaded, most likely by contamination from the uterine exudate. One cow showed tubercular abscesses in the ducts. Invasion of the ducts has been referred to by many observers: Fröhner and Zwick, Hutyra and Marek, Williams and others. When infection takes place per continuitatem from a tubercular perimetritis, rectal examination may reveal the identity of typical tubercular peritonitis with the formation of tubercles, or it may be of the nature of fibrous adhesions passing between the uterus, uterine horns, ovaries, or broad ligaments. Williams indicates that the absence of abscesses in the adhesions helps to differentiate tubercular perimetritis from the adhesions caused by other uterine affections. The observations carried out here support Williams's view.

It is not uncommon for the serous surface of the uterus in tubercular metritis to remain free from infection. Williams suggests the probability of venereal infection in those cases especially if the oviduct is not involved. In one case of uterine tuberculosis postmortemed at this Institute the uterine lesions were younger than those in the lungs and peritoneum. The serous surface of the uterus was not involved, nor were the fallopian tubes. It is likely that a haemotogenous route would explain the infection in this case, especially as the herd bull could not be proved to be tubercular. Williams draws attention to the difficulty in definite diagnosis in the case of an enlarged, sclerotic uterus, free from adhesions. It is certainly difficult to differentiate the condition from sclerotic metritis and primary actinomycosis by rectal palpation, but the tuberculin test and microscopic examination of scrapings made from the uterine mucosa by means of a small curette, usually establish the nature of the lesion. These scrapings should for preference be taken from the horns of the uterus with a small curette curved slightly towards its extremity like a small uterine irrigation catheter. Cystic degeneration of the ovaries is a not uncommon concurrent condition with uterine tuberculosis. The cyst is usually the large type of Graafian follicle cyst which has been associated with nymphomania (Fröhner u. Zwick, Hutyra u. Marek, Williams). (Fig. 30, A.2.)

Macroscopical Appearance.

In the beginning the tubercular invasion of the uterine mucosa cannot be recognized macroscopically. It is only when the tubercles are somewhat established that they can be recognized. The volume of the uterine body and horns is not changed. Later the wall becomes thickened and firm. On palpation it may be possible to feel nodules in the wall by rubbing the fingers over it or by palpating between the thumb and fingers. On section, the mucosa is covered with a slimy mucous, later muco-purulent exudate mixed with caseous material. The tubercles are as a rule visible through the mucosa as greyish isolated spots which are raised above the surrounding mucosa, varying in size from a pin's head to a millet seed. On section, these nodules are greyish in colour and opaque. They are as a rule chiefly confined towards the epithelial surface. In older cases there is ulceration of the mucous surface. It presents a rough uneven appearance, greyish to greyish-yellow in colour with some reddish spots. (Fig. 56, A.2.) There is a greyish-yellow exudate, partly liquid, partly composed of necrotic and caseous flocculi. Tubercles .5 to 1 cm. in diameter project above the surrounding surface. On section of the wall, the tubercles extend throughout the depth of the mucosa or even into the zona muscularis. Many of them are caseous or calcified.

It is most common that both horns are equally involved, but asymmetrical horns are also met with.

When the serous surface of the uterus is involved in the tubercular process there are adhesions to neighbouring organs, the broad ligaments, the ovaries, the fallopian tubes, or the mesosalpinx. Frequently one finds typical tubercles in these adhesions.

Histology.

The tubercle bacilli appear to enter the mucosa by penetrating between the epithelial cells of the uterine glands or of the mucosa between the glands. Ludolph states that the tuberculous process begins in the neighbourhood of the mouths of the glands. Altenbrun and Fischer maintain that the uterine mucosa is first attacked, while Fischer believes that invasion also takes place through the uterine glands. It appears more than likely that the bacilli can invade any part of the uterine epithelium, the glandular epithelium and the epithelium between the months of the glands, since one frequently recognizes the youngest tubercular processes under the epithelium in both these situations.

In the youngest lesions the overlying epithelium does not appear to be injured. Epithelial desquamation takes place only when the tubercles are well established. The progress of the tubercles in the uterus and in the fallopian tubes appears to be identical. The tubercle begins as a small local centre underneath the epithelium, either of the gland or mucous surface. There is round cell infiltration around epitheloid cells and later giant cell formation. (Figs. 31, 32, 33, A.3.) Neutrophiles can also be recognized, but not nearly so frequently as the round cells. These small tubercles develop to invade the stratum cellulare and the epithelial covering. The epithelium becomes desquamated and removed, leaving an ulcerated surface of granulation tissue; between these superficial ulcers normal epithelium may still be present, or a local stratification may be observed. The increase in the size of the tubercles and their coalescence, which frequently occurs, cause a diminution in the number of uterine glands or the closure of their exit into the cavum uteri. Some of the glands are filled with cell débris and round cells, which undergo disintegration. (Fig. 34, A.3.) The glandular epithelium also undergoes change. The cells show local or diffuse desquamation. They become removed totally or partially so that the tubercle opens into the gland lumen. In this way the glands disappear until their remnants only can be recognized in the deeper layers of the stratum glandularis close to the muscular zone. Owing to the closure of some of the gland mouths and the continuance of more or less normal epithelium towards the fundus, the glandular secretion accumulates and gives the glands a cystic appearance. The epithelium of these cystic glands varies from cubical to flattened cells. In the older tubercles one recognizes disintegration, caseation, and calcification (Fig. 35, A.3.), surrounded by a zone of round cells in which giant cells are to be seen.

The surface of the mucosa is covered with a layer of necrotic caseous structureless material and purulent exudate.

When there is extensive change in the mucosa, the tubercular process may invade the zona muscularis either through the lymph or blood-stream, according to Aschoff and Frei. Kaufmann describes a similar extension to the myometrium in the human female. The invasion of the myometrium is favoured by pregnancy and during the puerperium. Tubercular perimetritis is similar to the lesions caused by tubercular invasion of the peritoneum in other situations.

Cervical tuberculosis would appear to be of rare occurrence. Williams described such a case. Frei apparently has not encountered this condition. Williams says the uterus and oviducts were involved. The lips of the cervix were swollen and hard. The lesion in the cervix was closed with no discharge, but there was the usual tubercular discharge from the uterus. Kaufmann says that the cervix is rarely involved in uterine tuberculosis in women. The condition has not been observed in the post-mortem examination of tubercular cows at this Institution, although tubercular metritis and tubercular invasion of the ducts of Gaertner have been seen in one case.

Prognosis.

The prognosis is hopeless inasmuch as the cow affected with tubercular metritis is an unfit companion for her stall mates and also for the stud bull. She is suffering as a rule from open tuberculosis and is a source of danger, not only to the stud bull and to her companions, but to humans. The tail and hindquarters are contaminated with tubercle bacilli, and it is easy to realize the ease with which the milk can become contaminated during milking, assuming that the udder has not already been invaded.

Sterility is a common sequel to tubercular invasion of the uterus. In view of the work of Wester in relation to spermalysin and spermatoxin, it is easy to understand that the spermatozoa cannot long survive the pathological exudate, should they reach the uterus. In less severe cases early abortion may take place as a result of failure of the embryo to become embedded (Frei). Should the embryo become embedded, abortion may take place as a result of the extension of the tubercular lesions in the uterine wall interfering with nutrition of the foetus. Should the foetus go through its normal gestation period in a tubercular uterus, there is the possibility of a congenital infection (Schlegel).

No treatment should be adopted. In uterine tuberculosis the danger of spread to humans and to the herd is a very real one. When the disease has been diagnosed the animals should be destroyed.

(5) CALCIFICATION OF THE COTYLEDONS AND FOETAL PLACENTA.

Calcification of the cotyledons and the foetal placenta has been observed in two cases. The after birth was retained in both. The adhesion between the maternal and foetal placenta was very firm, so that considerable patience was necessary in removal. The foetal membranes were removed in each case thirty-six hours after parturition. In the meantime a pessary containing 2 drams each of iodoform and boracic acid had been inserted into the uterus. Practically all the cotyledons were involved. Granules of calcareous material were withdrawn with the villi of the foetal placenta. These granules were irregular in shape and varied from 1 mm. to 5 or 6 mm. in size. They were firm and greyish-yellow in colour. They were fairly evenly distributed over the surface of the foetal placenta. The surface of the cotyledon was gritty to the feel and markedly pitted when the foetal placenta was detached. The smaller cotyledons were much more involved than the larger ones. The surface of many of them felt hard and gritty throughout.

Calcification of the cotyledons has been described by Williams and Zschokke. Williams states "that the cause was not clear. It was possibly the result of interplacental haemorrhage which stopped short of placental dehiscence and embryonic death. Had parturition occurred retained foetal membranes with necrosis of the cotyledons would probably have followed." Zschokke states that the calcification begins in the vessel walls of the uterus following a chronic metritis.

The history of one of the cases described here is unknown. The second case was that of a cow 4½ years old, which had been maintained for three years on a diet low in calcium but moderately high in phosphorus. (See animal 882 in the paper "Minimum Mineral Requirements of Cattle," Theiler, Green and du Toit.) She had had her first parturition—dead twin calves—14 months previously. After this parturition, the afterbirth was retained and a moderately acute metritis supervened. No calcification of the cotyledons was noted after the first parturition. She came into oestrum two and a half months later and conceived at the first service. She calved normally, but the afterbirth was retained. A moderately acute puerperal metritis followed, from which she recovered slowly.

Examination of the genitalia 32 days after the last parturition showed that the involution of the uterus was almost complete. It appeared to be normal in consistency. The middle uterine artery on the right was definitely enlarged, firm, and thick-walled. The enlargement was local, extending along the vessel for a distance of about 8 to 10 cm. from its uterine extremity.

At the moment of writing it is only two months since parturition. The cow has not yet shown oestrum, but it is intended to have her again served when the opportunity presents itself.

The chemical analysis of the calcareous material taken from the placenta was carried out by the Chemical Division of the Institute. Dr. H. H. Green says:—"The 'gritty' particles were dissected as free as possible of adherent flesh, and then ashed to remove organic matter. Composition of ash—CaO 56 per cent., P_2O_5 40 per cent., Carbonate, present. This ratio of CaO to P_2O_5, together with the sum of CaO and P_2O_5 at 96 per cent., indicates that the mineral matter is chiefly calcium phosphate, with only a small amount of calcium carbonate. For all practical purposes the mineralization of the cotyledons can therefore be regarded as ossification or deposit of bony material."

Histological examination of the foetal placenta showed the presence of several calcified areas. These areas were mostly irregular in shape; a few, however, were rounded with concentric ringing. (Figs. 28, 29, A.3.)

DISEASES OF THE FALLOPIAN TUBES.

Pathological changes in the fallopian tubes are found in a very large percentage of cows which suffer from permanent incurable sterility. It is doubtful whether salpingitis occurs with less frequency than pathological changes in the ovary. Williams states that 15.3 per cent. of cases examined for genital diseases showed pathological changes in the fallopian tubes. The study of the genital affections of cattle in South Africa has indicated the important position which salpingitis takes amongst the pathological conditions which interfere with reproduction. Tubal pathology is no less common here than it is in the United States.

Tubal affections do not occur independently. They are due to secondary invasion from some existing pathological lesion in the genital tract or in the peritoneum. When a history of affected cows is available there is evidence of a puerperal metritis, a cervicitis, or both, following on placental retention, difficult parturition, or parturition injuries. In herds where contagious abortion is known to exist, salpingitis very frequently follows the metritis of abortion. Nielsen believes that the adhesions found inside and outside the fimbriated extremity of the tubes are the result of parametritic extension rather than from extension from the tube itself. The frequency with which parametritis is found associated with pavilionitis supports this view. However, there is little doubt that the majority of tubal affections ascend from the uterine extremity of the tube. Tubercular salpingitis is quite frequent when tubercular lesions occur in the peritoneum. In this case infection of the tube occurs from the ovarian extremity. In other cases infection occurs from the cornual extremity as a result of extension from the uterus. Salpingitis is very commonly met with in herds amongst which contagious abortion and infectious vaginitis are intense and widespread, and in which desirable hygienic conditions are not maintained. It is the rule for farmers in South Africa to treat cows which abort by uterine irrigation, strong irritant disinfectants being used. In this country contagious vaginitis was looked upon by many as the most fertile cause of sterility amongst cattle, and as such it received the therapeutic attention of dairymen. It was treated in the most drastic manner with strong irritant disinfectants, with little aseptic precautions, with the result that it was quite a usual occurrence to find treated herds suffering from an acute vaginitis and cervicitis as a result of the drastic measures adopted to overcome the disease. It would appear that contagious abortion or infectious vaginitis was not the direct cause of salpingitis, but that the invasion of the salpinx was the result of infection due to secondary organisms conveyed during treatment. It is not difficult to imagine the ease with which the tubes can be invaded in a paretic uterus, filled with disinfectant and fluid which is introduced into the uterus through a not too clean irrigating apparatus. It is not contended that irrational treatment of uterine affections is the only cause of salpingitis. It is observed in cows that have never been treated, but unsuitable treatment of infectious vaginitis, metritis and retained afterbirth renders the possibility of tubal infection far more likely.

In the United States salpingitis would seem to be a common occurrence in heifers (Williams). This does not appear to be the case in this country. Only one case has been observed. The subject was an eighteen-months old Friesland heifer which had never been served. When 15 months old, the heifer began to show enlargement of the mammary gland, which became so enlarged that she had to be milked. Clinical examination revealed a non-pregnant uterus somewhat enlarged and flaccid with cystic ovaritis, hydrosalpinx, and cystic pavilionitis. (Fig. 47A, B, A. 2.) With this one exception, pathological changes in the tubes have been found in cows which have been bred. Williams has observed salpingitis in herds in which the disease could be traced to the bull without the incidence of an intervening pregnancy.

The bacteriological flora of the fallopian tubes is at present being investigated at this Institute, but the study has not preceeded sufficiently far for publication. In America the organisms most frequently found are a streptococcus of the viridans group, *Staphylococcus albus* and *aureus*, an organism resembing *B. abortus* (Gilman) and *B. pyogenes* (Beaver, Boyd, Fitch). *Streptococci*, *Staphylococci*, *B. coli*, *B. vitulinum*, and Bang's bacillus have been described in the normal tube by Find. Brandt found *Staphylococci* and *M. tetragenus* in the fallopian tubes of normal cows and bacilli in addition in pathological tubes. Hundsberger found *Staphylococci*, *Diplococci*, *Micrococcus tetragenus*, *B. coli*, and short rods in pregnant animals as well as non-pregnant animals. He, therefore, concludes that the presence of these organisms does not necessarily cause sterility. Many cases of old-standing hydrosalpinx would appear to be entirely sterile. During investigations into the bacterial flora carried out by Mr. Martinaglia, of this Institute, he describes having isolated *S. aureus*, *S. citreus*, and *S. albus from* one case, and a small haemophilic bacillus from another. Five others were, however, negative.

The diagnosis of tubal disease is of much economic importance inasmuch as clinical recognizable changes, bilateral in nature, are incurable. Examination of the tubes throughout their length requires considerable practice. Richter says that under normal conditions rectal palpation of the tubes is very difficult and often impossible. He maintains that if one finds an easily palpable tube at a rectal examination the diagnosis salpingitis is justifiable. However, to one experienced by frequent clinical examination, and with the possibility of making post-mortem examination to confirm his intra-vitam findings, tubal palpation becomes easy unless the tubes are surrounded by fibrous adhesions in the meso-salpinx. Williams gives a techinque for examining the tubes in detail. Palpation is begun at the fimbriated extremity and continued towards the ostium uterinum. It is readily admitted that the method gives a possibility of accurate diagnosis of changes in the tubes in most cases, but there are some cases in which the broad ligaments are elongated and the uterus somewhat enlarged as a result of many pregnancies, where it has been found more convenient and expeditious to examine the tubes from the cornual extremity. The ovary is first sought per rectum; it is then placed in the fingers of the other hand introduced into the vagina. The uterine cornu is then traced to its apex. The fixing of the ovary renders this manipulation extremely easy. From the apex of the horn the tube is traced to the ampulla with the thumb, first and second finger, and the examination completed by palpating the fimbriated extremity.

Careful palpation of the tube and its fimbriated extremity reveals minute pathological changes. Isolated cystic enlargements, 3 to 4 mm. in diameter, can be detected; adhesions on the inner and outer surfaces of the ovarian pocket can usually be felt. There are cases, however, where one finds micropathological changes in the absence of palpable macroscopic change. In those cases the tube is somewhat enlarged either locally or diffusely, but the finest tactile sense cannot detect the change at a clinical examination. In these cases a tentative diagnosis can usually be made by concurrent symptoms. Williams has pointed out that tubal disease is very frequently associated with cystic degeneration of the corpus luteum as a result of infection of the ovarian crater after ovulation. This cystic degeneration of the corpus luteum of the last interovulation period prevents normal physiological atrophy of the corpus luteum. This results in cessation of oestrum or irregularity of the interovulation periods, which are usually abnormally long. After some time oestrum will again occur, that is, when the lutein tissue in the wall of the cyst has become so atrophied by the internal pressure of the slowly developing cyst that it no longer inhibits oestrum. The opposite ovary then ovulates and the corpus luteum resulting is also retained and undergoes cystic degeneration in its turn, to follow the same path as its predecessor in the opposite ovary. It is not an unusual thing to see a large cyst with a trace of lutein tissue in its walls in one ovary and a corpus luteum with a developing central cyst in the opposite ovary. One sometimes finds three corpus luteum cysts in the ovaries, two in one and one in the other. (Fig. 26, A.2.) The lutein tissue in their walls clearly indicates the age of the corpora lutea. The oldest one, or that of the third last interovulation period, has a thin complete or interrupted band of yellow tissue, that of the second last interovulation period has a somewhat thicker band of lutein tissue, while the corpus luteum of the present interovulation period may show a small central developing cyst or it may be normal in appearance. If the case can be followed carefully, it may be possible to detect these changes in the ovaries. It is fairly safe to diagnose salpingitis in the absence of palpable lesions in the tubes if there is a history of placental retention, metritis, or cervicitis, followed by cessation or irregularity of oestrum with long interovulation periods. Failure of the corpus luteum to undergo the normal physiological atrophy after the eleventh or twelfth day of the interovulation period must be looked upon with suspicion in the absence of palpable pathological lesions in the genital tract or ovaries of sterile cows. Abeyance or irregularity of oestrum with long inter-ovulation periods is also observed in cases of functional sterility due to obesity produced by unsuitable diet and insufficient exercise, but careful investigation of the ration fed and the environment under which cattle are kept will help to exclude functional sterility. The history of the animals in these cases is of the utmost importance.

SYMPTOMS.

In the early stages of the disease palpation does not as a rule lead to definite diagnosis of salpingitis, since the tubes are not much enlarged. Usually the first symptom which can be diagnosed at a clinical examination is the presence of adhesions in the funnel-shaped extremity or adhesions between the free border of the fimbriated extremity and the ovary. In very acute inflammation the three coats of the tube may be involved, infection extending from the mucosa to the serous coat. Then one may find adhesions between the border of the mesosalpinx and the neighbouring organs. (Fig. 15, A.2.) As the disease becomes chronic, palpation reveals enlargement of the tube. At first the enlargement is local and is most frequently confined to the ovarian extremity. Enlargement may, however, occur at any point in the tube. The enlargements may be so arranged that the tube feels like a row of beads. (Fig. 26, A.2.) There may be only one or there may be several dilations along the course of the tube. In other cases the tube is diffusely enlarged from the uterine cornu to the fimbriated extremity. (Fig. 5, 12, A.2.) The tube becomes elongated and tortuous. Towards the ampulla it not infrequently forms several coils in the mesosalpinx, which appear as a fairly large cyst with an irregular surface. On palpation per rectum the dilated tube, whether the dilation be local or diffuse, is firm to the feel, but compressible on pressure; fluctuation cannot be detected. The dilations vary in size from a single cystic enlargement, a few mm. in diameter, to a diffuse enlargement along the whole length up to 1 to 1½ cm. in diameter. In advanced cases of salpingitis the pavilion is always involved. The first palpable symptom of invasion of the pavilion is the presence of adhesions in the funnel or around the attachment of the free extremity with the ovary (Williams). As the disease advances, the adhesions become more extensive until the pavilion becomes entirely closed, either by adhesions within the funnel itself or by adhesion of the free border to the ovary. In the latter case, the ovary is frequently encapsuled by the pavilion of the tube and the ovarian pocket so that it becomes difficult to palpate. (Fig. 51, 53, A.2.) The ovary can as a rule be easily felt, but definite diagnosis as to its condition is difficult owing to the encapsulation within the ovarian pocket and fibrous adhesions to neighbouring organs. Sometimes the ovary is not encapsuled, the free border of the pavilion is just attached to it. When the pavilion forms a closed sac, it always becomes filled with exudate, which on accumulation forms a cyst. (Fig. 26, 29, A.2.) The cystic enlargement may vary in size from 2 to 10 cm. in diameter. The cyst of pavilionitis is characteristic and not easily confused with a cystic ovary. It is a soft cyst, the contents not entirely filling up the capsule, which is thin-walled and tough. The swelling is quite independent of the ovary, not a part of it.

On post-mortem examination the enlarged tube of hydrosalpinx is, as a rule, thin-walled and whitish in colour. On opening, it contains an opaque, greyish fluid with some small flocculi, or the liquid contents may be quite clear. On close examination of its cross-section, it is possible in some cases to observe the enlarged and elongated mucous folds occluding the tube and forming septa between the cystic dilations. The contents of the cystic pavilion are also greyish opaque liquid with some flocculi, or the liquid may be clear. In one case the contents of the pavilion were markedly bloodstained and contained brown pigment, apparently the result of haemorrhages from the wall of a Graafian follicle which had opened into the pavilion (haematosalpinx). (Fig. 34, A.2.) There was no trace of tubal pregnancy.

In acute cases of salpingitis, where occlusion of the lumen of the tube occurs early as a result of swelling of the mucous folds, accumulations of pus occur. Local abscesses in the tube have not been observed. It has always been uniformly distended with pus. Sometimes the swelling is slight and the walls appear thickened. In other cases, however, where the wall is much distended, it is thin-walled and may even show fluctuation. In pyosalpinx the pavilion may also be involved. Pus accumulated in the closed extremity forms a soft abscess which is encapsuled in the thin-walled but tough membrane of the fimbriated extremity. There would not appear to be much tendency for the abscess to rupture. It is extremely difficult to make an intra-vitam diagnosis between hydrosalpinx and pyosalpinx at a clinical examination. Adhesions between the border of the mesosalpinx and the neighbouring structures would appear to be more commonly met with in pyosalpinx than in hydrosalpinx. In the latter condition, however, tense adhesions are sometimes met with. (Fig. 36, A.2.) A case in which a mixed pyosalpinx and hydrosalpinx coexisted has been seen. The condition was bilateral. The pavilion and about a third of the ovarian extremity of the tubes contained pus, while the cornual extremity was distended with an opaque watery fluid. (Fig. 37, A.2.) The condition described by Williams, resulting in necrosis of the ovary, has not been seen. Diseases of the fallopian tubes are in the great majority of cases bilateral. In one case only has unilateral hydrosalpinx been observed. It produced an incurable sterility in a valuable ten-year old Friesland cow.

Salpingitis is almost always associated with pathological conditions in the ovary. The most common lesion is that of retained and cystic corpus luteum (Williams). Retention of the corpus luteum causes oestral abeyance, or irregular oestrum with long interovulation periods. The cyst as it grows larger causes atrophy of the lutein capsule until the lutein tissue present no longer inhibits oestrum, which then results from the ripening of the Graafian follicle in the opposite ovary, or sometimes in the same ovary. The resulting corpus luteum also undergoes cystic degeneration, so that cystic corpora lutea may be found in both ovaries. It is not unusual to find one ovary small, inactive, and indurated, showing no evidence of developing Graafian follicles. (Fig. 22, 37, A.2.) That such an ovary has functioned normally is evident from the presence of corpora albicantia and immature Graafian follicles on microscopic examination. It is not easy to say when cystic degeneration of the retained corpus luteum begins and how long it takes for degeneration of the gland to be so complete as not to inhibit oestrum. No doubt both periods are very irregular. Corpora lutea have been found macroscopically, showing marked or commencing cystic development at the end of the third week after oestrum, and cows have been under observation which have shown interovulation periods varying from two to eight months. It is not claimed that the cessation of normal oestrum may not be due to other causes, but since the cows suffered from salpingitis with retained corpora lutea, it is not unlikely that the retained and cystic corpus luteum was culpable. Another frequent ovarian lesion which is found associated with salpingitis is the cyst which is apparently developed from the unruptured Graafian follicle and which has been associated with nymphomania (Fig. 12, A.2.)

There is not much doubt that the ovarian pathology is dependent on infection of the ovary from the diseased fimbriated extremity of the tube at the time of ovulation. This hypothesis is supported by microscopic evidence in the absence of macroscopic changes in the tube.

Tubercular salpingitis is also a common occurrence in bovines. It is likely that the infection in this case most frequently descends down the tube from the peritoneum. Infection from the uterus, through the blood or lymph stream or from the peritoneal surface of the tube, would appear to be of rare occurrence (Frei). Gilman maintains that the source of infection is as a rule haematogenic, though he accepts the possibility of spread from the vagina, ovaries, uterus, and peritoneum. It appears that the fimbriated extremity of the tube is in the majority of cases the site of tubercular lesions prior to the invasion of the lumen (Fischer, Hansen, Johne, Rieck, Eber, Meyfarth, and Frei). Williams states that even in cases of severe uterine tuberculosis the oviducts are sometimes free.

On palpation the tube is irregularly enlarged, firm, and nodular. In cases of long duration the whole length of the tube is involved in the dilatation, or it may be confined to the abdominal extremity. The cases observed were all apparently of long standing, so that the whole tube and the uterus were invaded with tubercular lesions. There was a local tubercular peritonitis on the uterus and uterine horns, mesosalpinx, and meso-ovarium. The surface of the ovary also showed tubercular lesions. The fimbriated extremity of the tube showed adhesions to the ovary, but it was not cystic. On section, the lumen of the tube is occluded with yellowish brittle caseous contents. When the contents are scraped off, the lining appears yellowish and granular with numerous reddish spots. The tubercular invasion of the tube was in the cases observed bilateral.

HISTOLOGICAL EXAMINATION.

In the case of catarrhal salpingitis, the mucosa is the seat of initial changes. It becomes swollen as a result of accumulation of serous infiltrate and round-cell infiltration. There is a dilatation of the capillary vessels. The mucous folds become swollen, sometimes filling up the lumen of the tube. An exudate is thrown out into the lumen, which contains desquamated epithelial cells. There is a loss of cilia and fairly well-marked epithelial desquamation, so that the mucous folds may be partly stripped of their epithelial covering. Williams states that the epithelial desquamation begins on the apices of the mucous folds and gradually descends into the intervillus spaces. The folds, when stripped of epithelium, coalesce by the development of fibroblastic tissue, so that the lumen of the tube presents a cyst-like appearance, the vesicles being lined by epithelium. The coalescence of the folds or the swelling of the mucosa in the early stages causes occlusion of the lumen so that the exudate accumulates within the tube. In very acute cases the muscular coat may also show round-cell infiltration and a slight serous exudate. Hyaline degeneration with calcification of the tunica media of the vessels has been seen in the apex of the uterine horn, but calcification has not been seen in the tube. (Fig. 26, 27, A.3.)

When occlusion of the uterine extremity of the tube is complete, the exudate accumulates within the lumen and is passed out into the fimbriated extremity, which also becomes involved in an inflammatory process (Williams). The result is the production of fibrous adhesions across the fimbriated extremity and between the free border of the tube and the ovary or neighbouring structures. Thus the tube also becomes occluded at its upper extremity.

As the inflammation subsides and the tube has been fortunate enough to avoid occlusion, it is possible that complete regeneration of the desquamated epithelium occurs and it returns to normal. There is little doubt that this does occur in many cases where a slight catarrhal salpingitis only was present. It is difficult to imagine a metritis with atony of the uterine wall in which the uterine extremity of the tube is not also involved. Still, with rational treatment, most of these cases recover and again become regular breeders.

When the tube is closed at its proximal and distal extremity, the contents accumulate, resulting in a hydrosalpinx or a pyosalpinx. On microscopic examination, the pathological changes which take place in the tube from the acute catarrhal inflammation to the well-established hydrosalpinx can be traced. The histological changes during the acute or initial stages have been described up to the establishment of occlusion. In the early stages of hydrosalpinx, when the acute inflammatory phenomena have subsided, a cross-section of the tube shows the presence of closed vesicles due to coalescence of the mucous folds. (Fig. 38, 39, A.3.) In places the normal pseudo-stratified epithelium can be recognized, mostly devoid of cilia. In other places the vesicles are lined by flattened epithelium, apparently the result of pressure. All these cysts with flattened epithelium contain fluid. Other vesicles are seen, the walls of which are pushed together so that they become adherent, forming secondary vesicles. The epithelium can be seen undergoing desquamation and atrophy as the result of pressure, so that the membrana propria of neighbouring folds coalesce. In places there is active multiplication of tissue as shown by the presence of foci of round-cell infiltration. Fibroblastic tissue and newly-formed connective tissue are also evident. (Fig. 38, 39, A.3.) The membrana propria is in cases markedly thickened with these newly-formed tissue elements. In well-established hydrosalpinx it appears that coalescence of neighbouring mucous folds and the formation of secondary cyst-like vesicles are the result of the change in the tunica propria pressing upon the epithelium, causing atrophy, thereby allowing the newly-formed tissue in the folds to coalesce. In some sections scattered through the epithelium there are occasional eosinophyles with polymorphic nuclei. Another large cell with small acidophile granules and a round or slightly oval nucleus was sometimes seen.

As the lesions become older and the intratubal pressure increases from accumulation of fluid, the vesicles become larger and the epithelial lining throughout is flattened or cubical. Here and there the epithelial cells are vacuolated and their surface covered with exudate. (Fig. 43, A. 3.) It is difficult to say whether the vacuole formation in the epithelial cells is associated with secretion or whether it is due to regressive change. The mucosa shows well-marked oedema, apparently due to diffusion of the vesicular contents. (Fig. 41, A. 3.) The muscular layers as a rule in those cases of marked tubal distension show a very marked atrophy. (Fig. 44, 45, A. 3.)

In the case of pyosalpinx, the wall is as a rule thickened, while the peritoneal covering is the seat of fibrous adhesions. The mucosa is infiltrated with round cells and neutrophiles. The epithelial layer has disappeared. The lumen is filled with pus. Here and there one occasionally sees cyst-like formation from coalescence of the folds, but in old-standing cases these disappear.

In tubercular salpingitis, as already stated, the invasion of the tube would appear to begin most frequently from the ovarian extremity, the fimbriated extremity being the first part to show tubercular lesions. The tubercle bacilli enter through the epithelium covering the mucous folds. The propria becomes the seat of typical tubercles, showing round cells and epithelioid cells, with giant cell formation, which gradually extend. Fischer and Richter describe round-cell infiltration of the mucosa and epithelial desquamation which has been referred to by Frei as probably an earlier stage. Gilman also describes profuse leucocytic infiltration and some epithelioid cells.

The folds become swollen and the epithelium rapidly disappears. The apices of the folds appear to be first involved, as they do in the case of catarrhal inflammation, so that one can recognize the advance of tubercular lesions towards the periphery. Ulcers are also recognized. The lumen of the tube is filled with exudate containing granular débris. With the advance of tubercle formation and swelling of the mucous folds the lumen of the tube becomes entirely occluded as a result of coalescence of the swollen folds and the tube becomes very much increased in size. Towards the muscular wall in the deeper portion of the mucosa, cyst-like cavities lined with epithelium can still be recognized, but these, too, gradually disappear from pressure of the growing tubercles. When the tube is very much enlarged, there is well-marked atrophy of the tunica muscularis.

One recognizes, in old cases, caseous degeneration and calcified areas in cross-section.

The tunica muscularis may also show the presence of tubercles so that it may be almost entirely destroyed. The tunica serosa in old-standing cases shows an increase in formation of connective tissue. There is perisalpingitis present.

PROGNOSIS

The prognosis of bilateral salpingitis which can be recognized by rectal palpation appears to be hopeless. The pathological lesions which such tubes reveal at a post-mortem and histological examination are incurable. It is possible that many cases of slight catarrhal inflammation, which are concurrent with or a sequel to metritis, recover spontaneously. The possibility of frequent tubal infections associated with metritis cannot be questioned. Especially is this the case in a paretic uterus during the puerperal period. Still, when suitably treated, many of these cases recover and again become regular breeders.

TREATMENT.

With our present knowledge, preventive measures only are likely to be of any use in the treatment of the condition. Therapeutic measures adopted have not been satisfactory. It is known that salpingitis is secondary to affections of the cervix and the uterus, such as are associated with metritis, contagious abortion, placental retention, parturition injuries, and irrational measures employed to overcome infectious vaginitis.

Williams points out the prevalence of tubal diseases in heifers, and suggests that it is probably referable to unsuitable hygiene during calfhood, the infection remaining dormant until sexual maturity has been reached.

Williams further points out that an infection which may produce salpingitis may be carried by the bull and introduced during coitus; that such an infection may not prevent conception, but may cause salpingitis during pregnancy.

It therefore becomes evident that breeding cattle and their offspring should be kept under ideal hygienic conditions if a high-fertility record is to be maintained. Further, cows should be watched carefully for anything pathological during the puerperal period. Such pathological lesions should receive immediate attention. Sex hygiene should be advocated. Cows showing any suspicion of genital infection should not be allowed to copulate until the nature of the lesion has been diagnosed. How often does one find a cow with a muco-purulent or purulent discharge from the womb being served by the stud bull, who thereby runs the risk of spreading the infection. It is notorious that in this country the prevalence of sterility in cows is proportional to the degree of hygiene maintained in the byre and calf-sheds and to the efficiency of the methods adopted in overcoming diseases of the genital tract.

Treatment of tubal disease must be expectant. If it does exist concurrently with cervicitis and metritis, these conditions must be treated by modern methods. The danger of irrigating a paretic uterus has long been recognized. Modern methods have shown that treatment with pessaries of iodoform and bismuth subnitrate, or iodoform and boric acid (Williams), charcoal sticks (Hoffman), carbo-medicinalis (Klein), and protargol sticks are more compatible with recovery and subsequent fertility than uterine irrigation. Chronic uterine lesions can be treated with saline injections or saline and Lugol's iodine in solution, rivanol solution, etc. Should a small quantity remain in the uterus, it is not likely to pass into the tube, especially when the wall of the uterus has more or less regained tone. If diseases of the tubes should be suspected, it would be unwise to express corpora lutea just because the interovulation period was abnormally extended. Thereby ovarian infection is rendered more likely. When uterine infections subside and the normal physiological relation of animals has been restored, the corpus luteum, unless already cystic, will probably undergo normal atrophy. In the absence of palpable pathological lesions in the tubes, expression of a retained or cystic corpus luteum can be tried. It has been pointed out that salpingitis is usually bilateral, but Williams has seen unilateral infections which were capable of diagnosis by rectal palpation, and recommends treatment by unilateral ovario-salpingectomy.

The literature on the treatment of salpingitis in human medicine indicates that there is a vast field for research in unspecific-specific therapy as indicated by Oppermann. Among the non-specific preparations used in human medicine are the following: milk, aolan, cascosan, novoprotin, omnadin, yatren-casein, yatren-vaccine, which are administered intracutaneously, intramuscularly, or intravenously. In veterinary practice this method of therapy, although tried in a limited number of cases by Oppermann and his students, requires further investigation before its use can be recommended as of universal value in the treatment of salpingitis.

Within recent years Stoss and Mai showed that it is possible to force air from the uterus through the tubes. A manometer showed that the necessary pressure was 160 mm. of mercury. The pressure gradually sank in the case of normal tubes, but it remained constant when the fallopian tubes were occluded in salpingitis.

Stoss states that it appears possible in cases of salpingitis catarrhalis to open the tube to admit the passage of spermatozoa in the lower animals, as has already been demonstrated in human gynaecology.

DISEASES OF THE OVARY.

INCIDENCE AND GENERAL CAUSATION.

Pathological Changes in the ovary are commonly concurrent with pathological changes in the tubes and the uterus. It would appear that diseased conditions of the ovary are rarely primary, since they are usually found associated with or as a sequel to inflammatory conditions in the peritoneum, uterus, or tubes. If a history of the case is available, the sequence of events indicates that the ovarian changes have occured subsequent to disease of the uterus and tubes. It is highly probable that ovarian pathology is a result of infection which invades the ovary as a result of extension through the fallopian tube, the portal of entrance being the ruptured ovisac (Williams, Albrechtsen). There is little doubt, however, that under certain circumstances organisms which invade the tube and its pavilion may enter through the unbroken surface of the ovary. Cystic degeneration of the Graafian follicle is sometimes encountered in a cow, which had suffered from puerperal affection followed by metritis chronica catarrhalis, a month or two after parturition in the absence of an intervening oestral period. It is possible, however, as Nielsen points out, that ovulation may have taken place in these animals in the absence of oestral symptoms.

The probable secondary nature of cystic degeneration is also pointed out by Nielsen, Albechtsen, Richter, and others. Nielsen, however, attributes the injury to the ovary, not to direct infection of the ovary itself, but rather to the effect of metritic toxins on the theca capillaries.

It is unlikely that ovarian pathology is the primary lesion which has developed when this condition is concurrent with lesions elsewhere in the genitalia. In the early stages of acute puerperal metritis the parenchyma of the ovary is not involved. The surface will probably show lesions when a perimetritis exists. The ovarian parenchyma becomes involved only after the infection has had time to spread through the tube to its fimbriated extremity, so that infection is brought in contact with the ovary. Clinical or macroscopical examination may not reveal the fact that the tube is the site of pathological change, but a histological examination leaves no doubt that the tube has been invaded.

In view of the fact that uterine pathological changes can be proved to precede cystic degeneration of the ovary, it is unlikely that there is a hereditary predisposition in certain breeds of cattle to the condition. Environmental conditions, except when associated with bad hygiene, do not seem to be a causative factor.

Pathological changes in the ovary have been observed clinically, macroscopically, or histologically in 66 per cent of the cows slaughtered, because of incurable sterility, during these investigations. Microscopic examination, however, demonstrated the fact that histological changes may be present in many cases which would have otherwise been considered normal. Large cysts either of Graafian follicle or corpus luteum origin and perioophoritis can readily be recognized at a clinical or post-mortem examination, but changes such as oedema, small cystic degeneration, and abnormal follicular atresia can be demonstrated only microscopically. In the absence of histological examination, there are many ovaries which would have been considered unchanged, which were undoubtedly pathological.

It is considered advisable to give some details of the measurements and the weights of the ovaries of normal cattle examined after slaughter at the abattoir, since there is no record in the literature of such data having been taken in this country. It is considered sufficient, however, to record the fact that much variation exists in the size and weight of the ovaries in normal cattle as pointed out by Hess, Zschokke, Ellenberger and Baum, Fitch, Sisson, Marquart, Hammond, and others. The length of the ovaries of normal cattle examined after slaughter varied from—left, 2.7 cm. to 4.8cm.; right, 2.7 cm. to 5.4 cm.; breadth—left, 1 cm. to 2.7 cm.; right, 1.3 cm. to 2.6 cm.; depth,—left, 1.8 cm. to 3.3 cm.; right, 1.5 cm. to 3.2 cm. Weight varied from: left, 3.6 gm. to 10.4 gm.; right, 3 gm. to 10.2 gm.

The ovary is in the majority of cases oval, but it is also liable to much variation. The presence of a corpus luteum may change the shape considerably (Marquart). It may be hour-glass shaped, roundly oval, or triangular. Richter draws attention to the fact that variations in the size and shape of the ovary must be considered when making a clinical examination. Richter also points out that the consistency of the ovary in the same cow may be different, when examined at different intervals during the oestrous cycle, as the result of periodic physiological change. Our clinical examinations support the statement of Richter. It is also necessary to observe the variation which may occur in the size of the developed Graafian follicle. It is easy to mistake a large Graafian follicle for a cyst. It is usual to consider that a developed follicle may reach a size of 1.5 cm. in diameter (Rubeli), 1-1.2 cm. (Zschokke), 1.3 cm. (Krupski), 1.6 cm. (Schmid). Richter indicates that the fully developed follicle may sometimes reach a diameter of 1.7 cm. Histological examination of some of these large cyst-like structures have shown them to be normal follicles. Nielsen indicates that the size of the normal follicle may vary considerably; that the diameter may reach 2 to 3 cm. and that the limits between follicles and cysts in respect of size is entirely indistinct. He suggests that follicles may grow to a large size for five to six weeks after parturition. This inclination has certainly not been noticed here. It is difficult to differentiate between a large cyst and a normal follicle at a clinical examination. The normal follicle, however, is more easily ruptured as a rule. The ripe follicle may rupture with gentle manipulation of the ovary, while the cyst as a rule requires some pressure; even thin-walled cysts do not rupture with the ease of a Graafian follicle.

Nielsen says that the symptoms of nymphomania must be present before a definite diagnosis of cystic degeneration can be made in the case of small cysts, but rightly points out that these signs may be expressed without palpable ovarian change.

The corpus luteum of oestrum reaches its greatest size about the tenth to the eleventh day of the interovulation period (Küpfer, Nielsen, Hammond, Zietschmann). It varies from 0.8 cm. to 2.5 cm. in diameter; according to Zietschmann, it may reach 3 cm. in diameter. Hammond gives an average size as 1.9 cm. It can be felt as a prominence on the surface of the ovary, usually running smoothly into the ovarian surface, sometimes, however, very prominent with a distinct neck. As it atrophies, the prominence disappears and the surface of the ovary again becomes smooth and its level uninterrupted. Corpora lutea (II) are not as a rule palpable at a clinical examination. One sometimes encounters embedded or central corpora lutea which are not palpable. These, however, can be considered as persistent corpora lutea, which are secondary to retention of exudate in the uterus in pyometra, hydrometra, or in metritis. The corpus luteum of pregnancy is also frequently embedded and level with the surface of the surrounding ovary tissue towards the mid-term and the later stages of gestation.

In functionally sterile females the ovary is usually in an inactive state. Occasional oestral periods are observed. The interovulation period is unusually long. During the interovulation period rectal palpation shows that the ovary is small and firm. The Graafian follicles are in a static state. No trace of them can be found on the surface of the ovary. The corpus luteum may be palpable in the other ovary, but usually it is not. It has become partially atrophied and embedded.

The cause of functional sterility has already been referred to in this article, and since it is intended to discuss the subject fully later, it is not considered necessary to go into detail now. It is considered sufficient to say that the static changes encountered in the ovary in functionally sterile cattle in the light of our present knowledge are primary; that the original lesion is in the ovary itself following on the metabolic change in these animals.

A systematic histological examination of the ductless glands other than the ovary in functionally sterile cattle has not demonstrated the presence of any constant lesion in these organs. This work, however, is at the moment incomplete and no definite statement based on a sufficient number of examinations can be made at present.

These static changes, resulting in reduced activity of the gland, are caused by obesity, inactivity (Nielsen, Williams, Marshall and Peel, Gibbons, and others), senility and poor condition (Richter, Marshall, and Hammond). These conditions are produced by irrational feeding, or lack of feeding, or feeding a ration deficient in protein, mineral salts, and green food, coupled with insufficient exercise; in other words, a ration and environment incompatible with high fertility. Drain on the system in heavy milkers also produces subfunction of the physiological activity of the ovary (Nielsen, Hammond, Dolder, and others).

In the absence of normal physiological activity, the ovary undergoes a partial atrephy, which may be considered an atrophy of disuse. The changes in the ovary associated with functional sterility are commonly encountered in South Africa, probably more so that in Europe. One is astonished at the number of sterile heifers and cows in well-bred herds of beef cattle in this country. Herds have been examined where 20 per cent. of the heifers have been sterile. In one Shorthorn herd 60 per cent. of the whole herd was sterile. In these herds other causes of sterility were excluded. An explanation may possibly be found in the fact that there are relatively few purebred herds in this country. Those that are maintained are usually in very high condition. They are regularly " shown " from calfhood. The best heifers are constantly maintained in " show " condition. So as to allow of maximum development, they are not put to the bull until they are past two years. Climatic conditions may also play a part. It appears likely that acclimatization of European cattle to South African conditions may take several generations. The great variation in seasonal temperatures and the lack of green food and roots in the winter months may not be a negligible factor. Periodic droughts experienced throughout the country reduce the condition of cattle and at the same time limit the amount of green food which can be fed. One is accustomed to associate these dry periods with anaphrodisia in a fairly large percentage of cattle which are in low condition.

RELATION OF THE CHANGES IN THE OVARY TO THE OESTROUS CYCLE AND OESTRUS.

Normal bovine females in South Africa show oestrum regularly throughout the year at intervals varying from 19 to 22 days. The period is somewhat longer in winter than during the spring, summer, and autumn. The duration of oestral symptoms varies from 17 to 24 hours. Under South African conditions periodicity of the physiological changes during the oestrous cycle are similar to those in Europe (Hammond, Küpfer). Silent heat, described by Küpfer, Strodthof, Nielsen, and others, would appear to be not uncommon in sterile cattle. It has been encountered in several cases of over 50 cows kept under observation for periods varying up to two years. Nielsen states that these heatless ovulations are exceedingly common in cows, and that they occur quite frequently during the first two or three months after calving.

The majority of sterile cows shows change in the periodicity of the oestrous cycle. This does not apply solely to those sterile females in which pathological change in the ovary can be demonstrated at a clinical or post-mortem examination. Females suffering from sterility due to pathological changes in the uterus and fallopian tubes,in the absence of macroscopic change in the ovary, also show irregularity in the oestrous cycle (if kept under observation for a long period of time). Many of those cases, however, show histological changes in the ovary. It is quite likely, however, that pathological changes in the uterus, in the absence of ovarian pathology, will be sufficient to cause disturbance in the oestrous cycle. Nielsen has pointed out that the post-parturition heatless period in normal cows is shorter than in cows suffering from endometritis. He further states that the heatless period is increased with the intensity of the endometritis. All observers agree that there is cessation of heat in pyometra and hydrometra, in which conditions the corpus luteum is retained.

Daily observations have been made on over 50 cows suffering from sterility caused by various morphological changes in the genitalia, and also on several cases of heifers suffering from functional sterility. Some of the females concerned have been specially kept for a period of two years to observe the relation of the changes in the ovary and uterus to the oestrous cycle. In Appendix V a record of the frequency of oestral symptoms in these animals is recorded, as well as the diagnosis in each case. From this table it is possible to compare the oestrous cycle of cows and heifers sterile from various causes with normal cows.

Females suffering from functional sterility as a rule show an oestral period with less exaggerated symptoms than normal females. In many cases the duration of oestrum has been somewhat shorter in these heifers than in normal cattle. The interovulation periods are unusually long, extending in some cases over a period of months.

A long interovulation period may be followed by a few oestral periods at normal intervals. Then another long heatless period may follow.

Animals suffering from acute endometritis do not show oestrum. When chronic endometritis occurs as a sequel, in the absence of palpable change in the ovary, the post-partum heatless period is prolonged, as indicated by Nielsen. The period varies with the intensity of the endometritis. When oestrum does appear in cows suffering from endometritis, it frequently occurs at abnormal intervals if observations are continued over a sufficiently long period and the animals are left untreated. Successful treatment influences the oestrous cycle; it quickly becomes normal.

Cystic degeneration of the Graafian follicle has in about 75 per cent. of cases an extraordinary influence on the oestrous cycle. At first the symptoms of oestrum become more intense and the oestral period is more prolonged. This is followed by a decreasingly short interovulation period. It would appear that ovulation does not necessarily occur—in fact, it rarely occurs—in these cases. No corpus luteum is developed. On this account Hammond states that the ripening of follicles proceeds unchecked and the animal becomes a nymphomaniac. When the cysts are well established, the animal may show continuous heat and take the bull at any time. If cows suffering from nymphomania are kept sufficiently long under observation, without treatment of any sort, in the case of large cysts the whole ovary becomes practically atrophied, only a thin shell of fibrous tissue remaining. At this stage the symptoms of oestrum become less exaggerated. The cow may not allow copulation, but she may still exhibit the inclination to mount other females. Attention is drawn to the fact that nymphomania may occur in the absence of ovarian change in a small percentage of cases. Further, cystic degeneration of the Graafian follicle may be present without symptoms of nymphomania.

Cystic degeneration of the corpus luteum or Graafian follicle cysts with lutein tissue in their walls and the retained corpus luteum of pyometra are characterized by symptoms of anaphrodisia. The lutein tissue present inhibits development of the Graafian follicle and ovulation. Interovulation periods in these cases of cystic degeneration may be prolonged for months. The cyst appears gradually to increase in size until only a thin capsule of lutein tissue is left in the wall of the cyst. The lutein tissue then no longer inhibits the development of Graafian follicles, and ovulution occurs. The corpus luteum which develops as a rule follows its predecessor and in its turn becomes cystic. In the majority of the cases observed during these investigations this pathological cycle was repeated. The interoestral period was abnormally long, but the interval varied considerably; in some cases it was weeks, in others months.

PATHOLOGICAL CONDITIONS OF THE OVARIES.

(*a*) Aplasia.

Congenital aplasia of the ovaries has been met with associated with hypoplasia of the genitalia in the case of "free martins." (Fig. 45 B, Appendix II.) Outside "freemartinism," it is apparently an exceedingly uncommon condition in female bovines. It is consequently of little practical importance as a cause of sterility.

(*b*) PERIOOPHORITIS.

Perioophoritis (Figs. 20, 32, Appendix II) is quite a common pathological condition of the ovary. It is secondary in nature. It is found associated with tubercular lesions in the peritoneum, especially when there is tubercular perimetritis or perisalpingitis. It it also associated with perimetritis and perisalpingitis originating from causes other than tubercular. It is found concurrent with salpingitis and pavilionitis, apparently the result of extension of infection from the fimbriated extremity of the diseased tube.

The lesion may be local, confined to a part of the surface of the ovary (Figs. 26, 32, Appendix II), or it may be diffuse (Figs. 20, 53, Appendix II). Bands of fibrous tissue extend between the surface of the ovary and the neighbouring structures, the extremity of the tube, the mesosalpinx, the broad ligaments, or the uterine horn. The whole ovary may be firmly encapsuled by fibrous tissue or concealed within the ovarian pocket, which is adherent to the ovary. When complete encapsulation takes place, it is usually difficult to palpate the ovary, as the tube, the mesosalpinx, the broad ligament, and the uterine horn are adherent in a mass of fibrous tissue.

In cases of this nature the ovary is frequently cystic, showing cystic degeneration of the Graafian follicle, but more frequently cystic corpus luteum. Not infrequently the ovary is atrophied and sclerotic. The condition has been described by Williams, Hess, and others.

Since it is of a secondary nature, and probably in the majority of cases is preceded by incurable pathological changes in the uterus and the tubes, perioophoritis as a separate disease plays a minor part in the causation of sterility. When it is extensive and bilateral, it prevents normal ovulation and entrance of the ovum into the tube. However, in our experience the tube has been the site of inflammatory changes sufficient in themselves to render such animals permanently sterile.

(*c*) NEWGROWTHS.

Neoplasms of the ovary in cows would appear to be of comparatively rare occurrence. Two cases only have been met with. One was in the nature of a cystadenoma, the other was a teratoma.

(I) *Cystadenoma of the Ovary.*

A cystadenoma was met with in one case of sterility. It could not be considered as the cause of sterility in this case, as there were pathological changes recognizable on macroscopic or microscopic examination in every division of the genital tract and ovary. (Fig. 40, Appendix II.) It was discovered only on microscopic examination of the right ovary of bovine No. 2112. The ovary in which it was observed is described in detail in Appendix I. The tumour was confined to the medulla of the ovary and did not appear on the surface. It was exceedingly small, oval in outline, about 2 mm. in diameter by 8 mm. in length. The lesion could not be seen macroscopically without a very close examination. Owing to its size, it presented little difference to the neighbouring ovary tissue, which in this case showed dilated blood-vessels and lymph spaces.

The histological examination shows the lesion to be well marked off from the surrounding ovary tissue by a connective tissue capsule. The whole tumour inside the connective tissue capsule shows gland-like formation with numerous irregular acini. (Fig. 54, Appendix III.) The acini are rounded, irregularly elongated or branched. They are for the most part lined with a cubical to low cubical stratified epithelium, in some places several cells high, in other places two to three cells high. Some acini show a single-cell epithelial lining. The epithelium lies on a basal membrane of connective tissue. The walls of the gland-like structures are composed of thick bands of fibrous tissue in which blood-vessels are fairly frequent. The lumina of the acini appear to be empty. Scattered here and there are gland-like tubules which do not present a lumen, being filled with epithelial cells, apparently the epithelial wall which has been cut at a tangent.

This variety of tumour appears to be of fairly common occurrence in the lower animals. It would appear that they sometimes reach an enormous size, even up to 90 Kg. (Kitt). They have been observed in cattle by Johne, Joest, Seubert, and others. The condition is apparently similar to that described in the human ovary by Aschoff as glandular cystoma.

(II) *Teratoma of the Ovary.*

The one case which came under notice, was a specimen sent from the Durban Abattoirs for diagnosis. (Fig. 59, Appendix II.) The history of the animal is unknown. The right ovary was involved. It was 3.5 by 2.5 by 2.4 cm. in size. The shape was oval and the consistency firm. There were four irregular yellowish-brown areas on the surface, apparently the remnants of corpora lutea of previous interovulation periods. The ovary had apparently ovulated. Towards the anterior pole there was a thin capsule through which bright yellowish contents could be seen. On section, practically the whole ovary was made up of yellow, crumbly, slightly adhesive material, showing admixture with white hairs. The hairs were not very numerous, but were diffusely dispersed. The contents were easily enucleated from the capsule from which several of the hairs appeared to grow. The inner surface of the capsule was otherwise smooth and greyish-white in colour. Towards the posterior pole at its widest part the capsule was .6 cm. in thickness. No macroscopic evidence of Graafian follicles was present.

The left ovary—2 by 1.4 by 1.3 cm.—showed a prominent corpus luteum menstruationis which filled practically the entire ovary. This ovary appeared quite normal.

The pathological change must be considered a dermoid cyst.

Neoplasms of the ovary, owing to the rarity of their occurrence, appear to be of little practical importance as a causative factor in sterility of bovines. Since the other ovary was normal, and both tubes were unchanged, it is quite possible that the cow had been fertile.

(*d*) OVARIAN CYSTS.

(I) *Small Cystic Degeneration.*

In a small percentage of the ovaries examined, changes have been recognized which are considered similar to the " Kleinzystische Degeneration der Ovarien " of Aschoff. It has been met with chiefly in association with pavilionitis with encapsulation of the ovary within the ovarian pocket or the fimbriated extremity of the tube. (Fig. 34, Appendix II.)

Macroscopically the ovary presents a moist oedematous appearance which may be local, confined to one portion of the ovary. There are usually several small cysts close together. They vary in size from a few millimetres to 1 mm. or less; many are microscopic in size. They appear to be confined usually towards the centre of the ovarian tissue. They have not been frequently recognized towards the periphery.

Microscopically the cyst wall is composed of connective tissue lined with a single-cell layer of epithelial cells. (Figs. 58, 59, Appendix III.) Theca cells or follicular epithelial cells have not been seen in the wall. The cilia described by Aschoff have not been seen. In the neighbourhood of the cyst the ovary tissue is always oedematous. The tissue is loose and the interstices filled with fluid.

The spaces thus formed are sometimes large, sometimes small. usually spindle-shaped, but also oval and irregular in outline.

(II) *Cystic Corpus Albicans.*

Macroscopically this condition has not been recognized. In a small percentage of the ovaries examined cystic degeneration of the corpus albicans has been observed microscopically. (Fig. 55, Appendix III.) The corpus albicans presents a central space which is filled with liquid, staining pink with eosin. Immediately surrounding the cavity there is a loose fibrous tissue wall which shows spaces filled with fluid. Towards the periphery of the body there is a fairly dense connective tissue wall of considerable thickness which is not infiltrated by fluid. The contrast in the depth of staining of the peripheral and central zones of the wall is very marked. In no case has there been an epithelial lining recognizable.

(III) *Cystic Degeneration of the Graafian Follicle.*

Cystic degeneration of the Graafian follicle has been described by Hess, Zschokke, Albrechtsen, Richter, Nielsen, Opperman, Frei, Williams, Hammond, and others. In fact, the veterinary literature of countries in which interest has been taken in studying the diseases associated with sterility in cattle is rich in references to this pathological change in the Graafian follicle.

This is by far the most frequently encountered form of cystic degeneration occuring in the ovary.

The relation between pathological changes in the ovary and inflammatory changes in the genital tract has already been discussed. It is considered that the ovarian pathology is secondary to diseases of the cervix, uterus, and the fallopian tubes. In this connection, the hypothesis of Albrechtsen and Richter can be supported. Williams says he cannot verify Albrechtsen's assertion that the disease follows inflammatory changes in the uterus. He, however, admits the possibility. He says that the endometritis accompanying nymphomania may be due to the influence of the ovarian disease.

The immediate cause of the changes is most likely infection which gains entrance to the ovary through the ruptured ovi-sac, the lymph-stream, or, in some instances, through the unruptured surface of the ovary, as indicated by Albrechtsen, Richter, Oppermann, Reinhardt, Stalfors, and others. Similar bacteria have been found in the ovaries to those which have been isolated from the tubes (Find), namely, *B. abortus* Bang, *Staphylococci*, *Streptococci*, *B. coli commune*. Fitch has isolated *Streptococci*, *B. coli*, *M. pyogenes*, and a rod-shaped gas-producing organism.

Investigations which are being conducted to ascertain the nature of the bacteriological flora of the pathologic genitalia of cows at this Institution, however, reveal the fact that by far the greater majority of these cysts are sterile. This, however, is no indication that the ovary had not harboured bacteria earlier, since the fallopian tubes in cases of old-standing hydrosalpinx are also often sterile. The hypothesis of Hess, that ovarian change is primary, appears very unlikely.

It is agreed by practically all observers that the cyst is caused by the failure to rupture of a Graafian follicle which has developed to maturity. Several cysts may be formed in the same way.

The cysts which develop may be multiple (Fig. 12c, 17B, 30B, Appendix II) or a single cyst only may be found (Fig. 12c, 30B, Appendix II). The multiple cysts are frequently embedded, none of them being superficial. They vary considerably in size and sometimes in shape. They are usually from .5 cm. to 2 cm. in diameter, or about the size of a fully developed follicle. They are usually rounded or oval in outline, occasionally half-moon or crescent shaped. There may be two or three of such cysts or there may be five or six present in each ovary. The affected ovary is increased in size and somewhat rounded. On palpation it is softer than normal and slightly compressible, but fluctuation as a rule cannot be demonstrated.

At a clinical examination a definite diagnosis is usually difficult, unless several cysts are present. The softness of the ovary and its tendency to become rounded, with the history of the case, however, helps the clinician. Simon and Hammond say that they have found small multiple cysts in the majority of cases of sterile cows examined. Our observations here indicate that such is also the case in South Africa. They are more frequent than single cysts. Multiple large cysts are, however, not infrequent. (Fig. 12c, 30B, Appendix II.) They vary considerably in size from 2 cm. to 5 or 6 cm. in diameter. There may be two or more of these large cysts present. The ovary is increased in size and lobulated. Fluctuation can also be demonstrated. Each ovary may reach a diameter of several centimeters. The cysts may be rounded, oval in shape, or half-moon or crescent shaped. The smaller cysts are pressed upon by the larger ones and in consequence tend to deviate from the usual rounded shape. The capsule of these cysts is thin, somewhat tough, and whitish in colour. It is often transparent. The septa separating the cysts are also thin. Most of the ovarian tissue may have undergone pressure atrophy. What remains is moist, and whitish-grey in colour.

A single large cyst is less frequently met with. (Fig. 33, Appendix II.) Such a cyst may be the result of coalescence of multiple cysts. The ovary is increased in size and fluctuates. It is rounded or oval in outline. Cysts 6 to 7 cm. in diameter have been seen. Richter has observed a cyst 13 cm. in diameter and 990 g. in weight. The cyst wall is thin and somewhat tough. The lining of the wall is smooth. Villi have occasionally been seen. (Fig. 70, Appendix III.) As a rule, little or no ovarian tissue is left. It has undergone atrophy.

The contents of these cysts is a transparent thin clear liquid, sometimes in the smaller cysts with a slightly yellowish tinge. Frequently it is slightly opaque and contains very small whitish focculi. It varies considerably in amount according to the size of the cyst. The cyst is always entirely filled with fluid. In 10 per cent. formalin the fluid quickly undergoes coagulation. It becomes jelly-like, greyish, and semi-transparent.

Chemical analysis of the fluid was done by the Chemical Division of this Institution with the following results:—

> Dr. H. H. Green's note on the analysis reads as follows:—
> "Analysis made on the fresh material 20 minutes after removal.
>
> Reaction PH. 7.8, i.e. distinctly alkaline; specific gravity at 20° C., 1.010; protein, 0.97 per cent.; non-protein nitrogen, negligible; chloride, expressed as NaCl, 0.38 per cent.; total solids (dry matter), 1.89 per cent.; ash, 0.92 per cent.; alkalinity of the ash per 100 c.c. = 3 c.c. N. acid; phosphorus, as phosphoric oxide, P_2O_5, 0.003 per cent.; inorganic phosphorus, negligible; reducing substances, negligible.

"This is only a proximate analysis similar to that on page 66 of Hess. Comparing with the data of Hess, it will be noted that the fluid is much more watery (only about a quarter of the total solids) and with only about 1/6th of the protein. Practically the whole of the N. present is there as protein. The ash, however, is just as high (.92 per cent. against .89 per cent.). The phosphorus is much lower and apparently in organic form."

Cystic degeneration of the Graafian follicle is frequently concurrent with nymphomania and the anatomical changes associated with it. It has, however, been pointed out that nymphomania may occur in the absence of cystic change in the Graafian follicle, and that cystic change in the follicle may be present in the absence of nymphomaniac symptoms. The symptoms associated with nymphomania and the anatomical changes which occur in the head, neck, and rump have been described in connection with diseases of the uterus. A small percentage of cows suffering from Graafian follicle cysts shows anaphrodisia. Schumann and Hieronymi state that in cows with cystic ovaries 70 per cent. are nymphomania and 30 per cent. show anaphrodisia. Hammond further states that anaphrodisia may occur in the case of single large cysts and nymphomania in the case of multiple cysts. His explanation is that the large cyst prevents the development of other follicles.

The association between metritis, salpingitis, and pavilionitis and cystic degeneration of the Graafian follicle has already been referred to. When cysts have been found in the ovary, the presence of pathological changes in the uterus or the fallopian tubes or both have been demonstrable either clinically, macroscopically, or microscopically. Perioophoritis is also a common occurrence.

It is unusual to find palpable cystic change in the ovary within two months after parturition, but usual after that time. One case, however, has been met with in which a valuable Friesland cow showed symptoms of nymphomania and bilateral cystic degeneration five weeks after parturition. Williams states that he has seen the disease 15 days after apparently normal parturition without clinical manifestations of metritis. Such a case is, however, probably exceptional. According to Albrechtsen, cysts usually appear fourteen days to one month after parturition or abortion. This, however, has not been our experience in this country.

Histological Examination.

The first literature on the histological appearance of ovarian cysts appeared (Zschokke) in 1900. He describes a connective tissue wall poor in blood-vessels in large cysts. In the small cysts, however, the specific cells of the zona granulosa and the theca interna could still be recognized.

The histological appearance of the cyst originating from a Graafian follicle depends upon the stage at which it is examined. The cyst is developed from a Graafian follicle which has apparently reached maturity, but which has failed to rupture. The first change takes place in the contained ovum, which undergoes degeneration (Wester, Zschokke). [Zschokke, Rubeli, and Simon have failed to identify ova in cysts originating from the Graafian follicle. Wester gives a photograph (p. 42) showing fatty degeneration of the ovum and degeneration of the cumulus oophorus as the initial stage in follicular atresia.] This is followed by degeneration of the cells of the cumulus oophorus. Hammond indicates that in follicular degeneration the granulosa is the first layer to break up. He says "it is not unlikely that, if the granulosa remains intact when the follicle fails to rupture, further increase instead of decrease in size takes place." The follicular epithelium in turn gradually degenerates, its inner layers of cells being the first to disappear. Cell débris may be seen in the liquid contents. In some cysts the layer of follicular epithelial cells inside the theca interna may persist for a considerable period. Sometimes it seems to persist as a permanent epithelial lining. (Fig. 60, 62, 71, Appendix III.) During this period of change in the follicular epithelium there is an apparent increase in the connective tissue in the theca, but theca cells can still be recognized. Some areas of hyaline degeneration in association with the vessels may be present. Bengisch describes hyaline degeneration of the interna in the early stages. The degeneration may be partial, or total degeneration of the theca interna may be present. He states that degeneration begins in this zone of the follicle to be followed by epithelial degeneration. Somewhat similar histological changes have been described by Kaufmann and Cohn in the human female, by Burghardt in the mare, and Simon, Fitch, and Wester in the cow. Eventually the change is complete. The follicular epithelium has entirely disappeared and the capsule is composed entirely of fibrous tissue in which some small blood-vessels may be present. The presence of lutein cells such as are recognized in the corpus luteum has not been seen in the wall of these cysts. Williams states there is never any lutein tissue in the cyst wall.

A second type of cystic change in the follicle has been described by Frei, Nielsen, and others. The follicle fails to rupture, but there is a development of lutein tissue from the zona granulosa and theca interna. This variety of degeneration is discussed later under cystic corpus luteum.

Fitch describes a connective tissue wall and a lining of small round or slightly oval cells several cells thick. The thickness, however, varied in different places. Hammond describes a cyst with a thin layer of normal granulosa cells. In another case he mentions the absence of a granulosa layer.

In our experience cystic degeneration of the follicle is always associated with local or diffuse oedema of the ovary. Bengisch says, in relation to this, the externa of the cystic follicle shows serous infiltration and the stroma in the neighbourhood is loose and atrophic.

Prognosis.

The prognosis of cystic degeneration of the Graafian follicle must always be considered serious. Since in our opinion it is secondary in nature, the prognosis depends upon whether the changes in the uterine mucosa or the tube are curable. The obstinate nature of cases of metritis of old standing is recognized. The hopelessness of resolution in established cases of hydrosalpinx is apparent. Many cases which are treated early before irreparable pathologic changes have occured in the uterus, the tubes, or the ovary itself, however, respond to treatment. In general, early treatment, which is begun before pressure atrophy of the ovarian tissue has extensively developed, is successful. In advanced cases of nymphomania, however, treatment is not hopeful. Recurrences are frequently experienced.

Hess's statistics indicate the apparent success with which he treated ovarian cysts by rupture. His diagnosis, however, is questioned by many well-known authorities, including Albrechtsen, Richter, and Nielsen. It appears probable that Hess in many cases was dealing with normal follicles instead of cysts. In view of the excellent results he obtained by ovarian treatment alone in sterility and the difficulty of making a clinical differentiation between small cysts and normal follicles, the doubt would appear to be justified.

In our experience cystic degeneration associated with nymphomania, with anatomical change in the rump and fore extremity, has failed to respond to treatment. Successful cases have been confined to cows treated early and in which there was no anatomical change. Williams, however, has had success in some cases of advanced nymphomania. Williams and Hagan say the prognosis is fair. The outlook for recovery is better when treatment is undertaken early. In old-standing cases where the disease has existed for one or two years the prognosis is bad but not hopeless.

Treatment.

In the treatment of cystic degeneration of the Graafian follicle the practice of Albrechtsen, Richter, Oppermann, Frei, and others has been followed. The cysts have been ruptured by pressure through the vagina. The left hand is introduced into the rectum and the ovary picked up. It is then placed in the right hand, which has been introduced into the vagina. Pressure is carried out through the vaginal wall. In addition, a course of bi-weekly intravenous injections of extract of corpus luteum has been given.

Large cysts whether single or multiple are easily broken down by pressure through the vaginal wall. Occasional cysts have been ruptured per rectum, but the practice has not been universally adopted. The small multiple cysts when deeply placed cannot be ruptured in this way without great pressure, which may cause irreparable injury to the ovary, or it is often quite impossible. In those cases it is better to wait and try again from time to time on account of the danger of injury to the ovary. The ovarian cyst knife described by Williams is useful in a large thick-walled central cyst, but it is of little value in the case of multiple small cysts. In fact, in this case its use is contra-indicated on account of the injury which puncture of each cyst would entail. Further, it is unlikely that four or five small cysts when placed centrally can be successfully punctured. In obstinate cases the withdrawal of the fluid with a syringe and suitable long needle has proved successful. Recurrences must be dealt with by rupture as they arise.

In addition to rupture of the cysts, uterine irrigation, as described under Endometritis, has been systematically carried out. As a stimulant, gentle genital and ovarian massage has been practised at weekly intervals during treatment.

Williams, Richter, Hammond, and others suggest removal of the affected ovary when the condition is unilateral. There is no doubt that cystic degeneration of one ovary causes sterility. Provided the ovary on the other side is not changed, it will act physiologically. This fact has been demonstrated by us in this country, in sheep in which one ovary has been removed in connection with investigations into sex production. In sterile cows, however, our experience has been that the majority show bilateral lesions. Peters has found in 74 sterile cows 42 with unilateral and 32 with bilateral ovarian change. Histological examination in these cases, however, may have shown a higher percentage of bilateral affection.

In cases which fail to respond to treatment ovariotomy should be performed as soon as a definite hopeless prognosis is made. It relieves the symptoms of nymphomania, allows the cow to maintain better condition, and prolongs the period of lactation for several months. Further, when the lactation period is ended ovariectomized individuals fatten more readily.

(IV) *Cystic Corpus Luteum.*

Cystic corpus luteum follows, in sequence of frequency, cystic degeneration of the Graafian follicle as a pathological change in the ovary. This form of degeneration has been described by Hess, Zschokke, Albrechtsen, Richter, Oppermann, Williams, Fitch, Küpfer, Elder, Frei, Williams and Hagan, and others. Cystic degenerative change has, however, been questioned by Nielsen, who maintains that cysts with lutein tissue in their walls are really Graafian follicle cysts in which a development of lutein tissue has taken place prior to the onset of degenerative change.

Hammond points out that at an early stage in the development of a normal corpus luteum (72 hours after the commencement of heat) there is a central cavity which becomes filled with a glairy albuminous fluid similar to liquor folliculi. He suggests that perhaps some of the corpus luteum cysts described by others are not pathological and that the majority represents a normal early stage in the formation of the corpus luteum.

Our observations lead us to believe that there is no doubt whatever that the corpus luteum undergoes cystic degeneration. When sterile cattle are kept under observation so that the changes in the ovary can be carefully recorded, the development of the cyst can be followed. Further, as stated elsewhere, when the other ovary ovulates—a phenomenon which takes place when the lutein tissue in the cyst wall has sufficiently atrophied so that it no longer inhibits the further development of Graafian follicles—the corpus luteum which is formed in the ovulation crater is usually retained and in time also undergoes cystic degeneration. Williams and Hagen have drawn attention to the likelihood of cystic degeneration of succeeding corpora lutea.

There is little doubt that this process of degeneration is pathologic. It is very rarely seen in pregnant cows, or non-pregnant cows which do not show pathological lesions of the genitalia. Williams has rarely seen small cysts in pregnant cows. Elder has seen one case. During our investigations a corpus luteum cyst has not been seen in an examination of a very large number of pregnant cows and normal non-pregnant cattle which had passed the first seven days of the interovulation period. When compared with our observations, Krupski would appear to have seen an extraordinary number of corpora lutea, varying from 10 to 20 days old, showing cysts in non-pregnant cattle. He has not seen the condition in pregnant cows.

As a causative factor, the condition must be considered secondary to pathological changes in the cervix, uterus, and tubes. Williams draws attention to the frequent concurrence of cystic corpus luteum and pavilionitis or encapsulation of the ovary within the ovarian pocket. Our observations at this Institution entirely support William's statement. (Fig. 20, 22, 26, 28, 36, 38, 41, 42, 43, 51, Appendix II.)

Here again the condition is probably due to ovarian infection by extension through the diseased fallopian tube. Williams says it appears justifiable to conclude that the disease is referable to an infection which enters the crater of the freshly ruptured follicle. A limited bacteriological study carried out at this Institution has shown that the cysts in old-standing cases are as a rule sterile. This, however, does not prove that bacterial flora from the tubes may not have been present at an earlier stage. Williams has found the presence of a streptococcus of the viridans group as the chief invader in cysts with lutein tissue in their walls.

Krupski has classified corpora lutea cysts as follows:—

(1) Those which arise, after bursting of the follicle, from the resulting corpus luteum. These produce no disturbance of the oestrous cycle.

This variety of cyst is possibly the small cavity which is encountered in the corpus luteum during the first days of the interovulation period. That it is not true degeneration of the corpus luteum has been pointed out by Hammond. Our observations support those of Hammond. It is maintained, however, that these cavities in the corpora of normal cattle are extremely rare after the first seven days of the interovulation period.

(2) Those resulting from the unruptured follicle, that is, Graafian follicle cysts with lutein tissue in their walls. According to Krupski, these cause interference with ovulation, but do not cause symptoms of nymphomania. Later when the lutein cells have disappeared ovulation occurs. As the cyst further develops, the interovulation period becomes shorter. There is a gradual transition to that condition of the oestrous cycle associated with the follicle cyst already described.

At a clinical examination the earliest stage of cystic degeneration cannot be recognized. It is only when the diameter of the cyst reaches about one-third that of the corpus luteum that it may be suspected. The corpus becomes soft and compressible and is usually somewhat larger than normal. The diagnosis is strengthened by the fact that the interovulation period is prolonged. As the cyst enlarges, which it usually does, a definite diagnosis can be made. The corpus luteum becomes enlarged, soft, and somewhat compressible. It undoubtedly contains fluid in its centre. The corpus no longer protrudes above the surface of the ovary. It may be level with the surface or somewhat sunken. The development of the cyst goes on at the expense of the capsule, which gradually becomes thinner and fluctuation becomes more evident, until a clinical examination fails to reveal the difference between the thick-walled cyst of corpus luteum origin and the thick-walled cyst of follicular degeneration. The ovary, which contains a well-developed cyst, is rounded or roundly oval in outline.

The cysts may vary considerably in size up to several centimetres. They do not appear, however, to develop to the same large size as the follicular cyst. Williams describes a large ovarian cyst, the orgin of which is unknown. He states it is not improbable that some of them may originate from the corpus luteum. This type of cyst has not been encountered during these investigations.

A history of the case is of considerable help in arriving at a definite diagnosis. These cysts are usually associated with infrequent oestrum and long interovulation periods. The interovulation period may vary from weeks to months. Nymphomania is not a sequel. Pressure on the ovary in the early stages causes total expression of the corpus, but in the later stages the cyst ruptures and the lutein tissue can be squeezed out from its wall. It has previously been stated that pavilionitis and salpingitis are frequently concurrent. This fact may be of significance in the early stages, when doubt exists, but it is not of pathognomonic importance, since it is also frequently present with cystic degeneration of the Graafian follicle. Some of the cases observed by us have shown the presence of a small false uterine plug, consisting of tough, tenacious, greyish material, almost similar to the uterine seal of pregnancy. The plug was usually present in cows which had had an interovulation period of two to four months prior to slaughter.

The time occupied in complete destruction of the corpus luteum by cystic degeneration appears to vary considerably. It can be approximately judged by the length of the interovulation period. What exactly influences the rapidity of degeneration it is difficult to determine. In some cases, as already mentioned, the interovulation period lasts a few weeks in other cases it extends to months. Williams states that degeneration may occur very rapidly, causing no material enlargement of the ovary. Sometimes the duration of the interovulation periods have been normal. He has seen oestrum occur in eight or fifteen days or it has been delayed 30 days or more. He says " it is the principal cause of irregularity in the oestrous cycle." This statement would appear to be undoubtedly correct when dealing with cows that have been pregnant. The chief cause of irregularity in the oestrous cycle of heifers in our experience is functional sterility due to obesity.

It becomes necessary to differentiate between the small cavities filled with fluid which very occasionally occur in the normal corpus luteum, especially during the first days of the interovulation period, and cystic degeneration of the corpus. The small central cavities resulting from failure of the lutein tissue to grow inwards and fill up the cavity do not interfere with normal physiological activity of the ovary. Cystic corpus luteum has an inhibitory influence on ovulation and oestrum. It is exceedingly difficult to determine whether all cysts showing lutein tissue in their walls are derived from corpora lutea, resulting from ovulation, or from unruptured Graafian follicles. That cystic degeneration of the corpus luteum does occur is certain, when sterile animals can be carefully studied. Sometimes the corpus luteum which develops in sterile cows after ovulation is soft. This may possibly be due to the fact that the ingrowth of lutein tissue only reaches a certain limit. The ovulation crater fails to become completely filled up. A central cavity which varies in size remains. The process of formation of lutein tissue is pathologic from the beginning. After a short time, the regressive changes associated with cystic degeneration takes place and the cyst gradually increases in size.

It is quite possible, as pointed out by Krupski, Frei, and Nielsen, that some of these cysts originate from the unruptured Graafian follicle. However, in each case they are responsible for a similar train of symptoms in the individual concerned. They are incapable of differentiation at a clinical examination once they have developed. They present the same macroscopical and histological appearance. Their significance as a definite pathological entity is undoubted.

Macroscopic Appearance of the Corpus Luteum Cyst.

The ovary, which contains a well-developed corpus luteum cyst. is as a rule rounded or roundly oval in outline. The cyst is usually thick-walled (Fig. 21, 42, Appendix II), but sometimes cysts are met with which may be comparatively thin-walled with a very thin band of lutein tissue in their walls. (Fig. 20, 36, Appendix II.) The lutein tissue in those thin-walled cysts may be confined to local areas (Fig. 20, 26, Appendix II) and not completely encapsule the cyst. However, the great majority of cysts observed by us showed a well-marked band of lutein tissue, varying from 1 mm. to 1 cm. or more in thickness. (Fig. 42, 43, Appendix II.) The lutein tissue may be normal lemon yellow to orange yellow in colour, but more frequently it is brownish, darker than the normal, or brownish-grey in colour. The cyst cavity contains a transparent or slightly yellowish fluid, which is somewhat viscid when rubbed between the fingers. The inner lining appears greyish or greyish-yellow in colour and is always smooth. The cysts vary from a few millimetres in diameter to several centimetres. They are not so easy to evacuate by pressure as the cyst developed from the Graafian follicle. After evacuation, if pressure is exercised on the ovary, the lutein elements in the cyst wall can also be squeezed out, unless it has more or less disappeared and is represented only by a very thin band. Elder says that cystic change may begin in several centres of the lutein tissue, it being possible to recognize several small cysts. In one case only have two small central cysts been seen in the same corpus luteum.

Histological Appearance.

The wall is composed of three layers which vary somewhat according to the stage of development of the cyst. In the case of a small cyst 3 or 4 cm. in diameter, the lining is of loose connective tissue which shows small spaces filled with fluid. This layer is very narrow. There is a thick middle layer of lutein tissue which cannot be differentiated from a normal corpus luteum. The fibrous elements of the inner layer can be seen running inward from the fibrous stroma of the lutein layer. The outer layer is a band composed of connective tissue, the bundles of which are interlaced and concentrically disposed, representing the tunica externa of the theca folliculi.

In cysts which have attained some considerable size three layers can still be recognized in the wall. The inner layer immediately surrounding the cavity of the cyst is composed of a well-marked zone of fibrous tissue which is somewhat loose in structure. The fibres are for the most part, however, arranged concentrically. Immediately surrounding the cavity there are fibroblastic elements present. There is usually some oedema of this zone. The middle or lutein layer is composed of a stroma of connective tissue in which some small blood-vessels can be seen. Lying in the fibrous stroma lutein cells are present, some apparently normal, others showing degeneration. The number of these cells appears to depend on the age of cysts or perhaps more correctly the size of the cyst. When a large cyst is present, there are fewer lutein cells than is the case with smaller cysts. The lutein tissue appears to disappear under pressure of the developing cyst. The elements of the outer layer also show a different appearance in the small and large cysts. One can recognize a transition between the apparently normal external thecal layer which surrounds the lutein layer in the case of very small cysts or commencing cystic formation, and a layer of dense connective tissue exceedingly poor in blood-vessels in the larger cysts.

Elder believes that the original change is that of hyaline degeneration which begins near the centre of the corpus luteum. Further, he states that degeneration may begin in several different places. Later, coalescence takes place as the areas of degeneration enlarge.

In the large number of ovaries showing cystic degeneration of the corpus luteum which have been examined large numbers of atretic follicles have been observed in the earlier stages of degeneration.

Prognosis.

In our experience the prognosis in these cases of cystic corpus luteum is unfavourable, since, as Williams indicates, it is so frequently associated with tubal pathology. Attempts at treatment carried out at this Institution have been usually unsuccessful. Those cases in which successes have been obtained have been early cases in which the cyst has not attained large size and in which the accompanying uterine affection has responded readily to treatment. That the cyst ruptures spontaneously has not been demonstrated here. In no case has spontaneous rupture been seen, nor does it appear likely when the nature of the capsule is considered. Williams and Hagan indicate the possibility of spontaneous rupture. They state: "So far as determined, the cystic disease of the corpus luteum has little significance beyond the one oestral period to which it belongs. Generally it appears to rupture within about three weeks, and disappears, a new ovi-sac ripens, and oestrum recurs at the regular time." Williams, however, later indicates that the prognosis in such cases is extremely serious. Hammond states: "Although cystic corpora lutea have been described by many investigators, it has not been shown that they are associated with sterility or any derangement of the cycle or of pregnancy." With this statement we cannot agree. It must be an extremely rare occurrence for cystic degeneration of the corpus luteum to occur except as a sequel to uterine, or tubal, disease; as such it requires careful attention. Large lutein cysts have been seen in which the ovary tissue had undergone atrophy and little more than a connective tissue capsule remained. It is most unlikely that such an ovary would again regenerate.

Krupski, cited by Frei, states that corpus luteum cysts are apparently without importance to the ovary when the volume of lutein tissue is not too large. A definite statement is, however, not made as to the volume of lutein tissue which will have an influence upon the ovary. He maintains that oestrum is not interfered with, that ripe follicles and corpora lutea of subsequent interovulation periods may also be present in the ovary. Further, the changes in the genital tract associated with oestrum may be concurrent with these lutein cysts.

Treatment

As a routine treatment, the uterus has been irrigated as described under the chapter on Endometritis. After this treatment had been continued for a couple of weeks, the corpus has been squeezed out by pressure through the vagina, or the cyst has been ruptured or, if rupture has not been possible, punctured, and the lutein tissue squeezed out from its wall. Williams' warning has made us careful to try and clean up the uterus before expressing the corpus luteum, in the hope of preventing further infection through the wound in the surface of the ovary.

When the uterus has cleaned up, a course of ovarian extract is given, at bi-weekly intervals, intravenously.

The possibility of curing the condition, when it is unilateral and concurrent with tubal disease on the same side, by ovariosalpingectomy cannot be disputed. This treatment has been recommended by Williams for unilateral salpingitis.

(V) *Persistence and Delayed Regression of the Corpus Luteum.*

Under this heading it is intended to include corpora lutea, which do not undergo physiological resorption after a normal interovulation period, an abortion, or parturition.

Persistence of the corpus luteum menstruationis has been described by most of the well-known authorities on diseases of the genitalia of the cow. Hess, Zschokke, Oppermann, Williams, Richter, Frei, and others have drawn attention to this condition as occurring frequently and causing sterility in the cow. Hammond, with a not too extensive material, considers he has seen retained corpus luteum as the sole cause of sterility in two cows. Richter has described persistence as an independent condition causing sterility in 12 per cent. of the cows which he examined. He saw it associated with endometritis in 5 per cent. of 204 cows which came under his notice. Albrechtsen and Nielsen question the theory of persistence of normal corpora lutea on the grounds that it is not a primary condition, but rather is secondary to inflammatory diseases of the genitalia. Richter agrees with Albrechtsen that persistent corpus luteum has been frequently erroneously diagnosed. He indicates the possibility of incorrect diagnosis by Schumann, who found somewhat more than 60 cows out of 1,600 examined with persistent corpus.

Regarding the causation of the condition, many theories have been put forward. If persistent corpus luteum does actually occur in normal cows, the cause of such persistence is unknown. Richter and Frei suggest the possibility of hormone influence. Our experiments have led to the belief that, if this is true, the disturbance comes from the ovarian or uterine hormone. A systematic examination of the ductless glands have up to the present failed to reveal any constantly occurring lesion outside the ovary itself.

Zschokke states that pathological retention is due to retained placenta and subsequent uterine infection. This contention is strongly supported by Albrechtsen, Nielsen, Williams, and Bongardt. Hammond doubts primary uterine infection as the cause. He says it appears uncertain whether uterine infection in itself causes persistence of the corpus luteum, or whether a persistent corpus luteum intensifies the uterine infection.

Zschokke connected the feeding of rye, malt, brewer's grains, and sugar-beet with persistence of the corpus luteum. According to Hammond, these foods increase the milk yield, and he suggests that the drain on the organism due to the increased milk secretion probably prevents the further ripening of follicles and therefore causes cessation of oestrum as seen in cases of corpus luteum persistence. He further suggests the possibility of these foods containing specific substances suitable for the nutrition of the corpus luteum and the mammary gland. Williams mentions that retention followed by cystic degeneration frequently occurs as a sequel to hypertrophied and haemorrhagic corpora lutea. (During the conduction of these observations, neither of these pathological conditions of the corpus have been seen.)

However, he has seen normal corpora lutea in normal cows tend to remain in the ovary without undergoing atrophy or degeneration. Williams draws attention to the doubt in correct diagnosis. He says it is quite possible that there may have been ovulation and silent heat. This may also be a possible explanation for the statements of Schmid and Kaltenegger that there is no histological difference in the corpus luteum which is retained and that of menstruation or of pregnancy. To eliminate the possibility of silent oestrum, careful and frequent clinical examinations of the genital tract must be made. The fact that oestrum does not appear is not sufficient evidence that ovulation has not occurred. It would be unwise to diagnose corpus luteum persistens in the absence of oestrous symptoms, without frequent clinical interpretation of the ovaries.

Nielsen believes that retention may follow an intense degree of endometritis, the direct cause being toxins produced in the uterus. In his experience there was much histological change in the retained corpus in the formation of connective tissue and sclerosis.

Our observations support the views of Albrechtsen, Richter, Nielson, Oppermann, and others, who look upon persistence of the corpus luteum as secondary to inflammatory lesions in the genitalia or to metabolic changes in the organism. It appears exceedingly doubtful if retention of the normal corpus luteum menstruationis occurs in normal animals in a state of physical fitness compatible with high fertility. The cases observed have as a rule been associated with a history of abortion, placental retention, and metritis.

Persistence has been rarely met with, as described by Williams, in an apparently normal animal. It is questionable, however, if a histological examination in these cases may not have revealed pathological changes in the uterus or tubes. The concurrence of persistence of the corpus luteum and lutein cysts has already been pointed out. Opperman has noticed the same concurrence.

Strodthoff and Nielsen do not connect the milder forms of endometritis chronica with persistence of the corpus luteum. Williams, Strodthoff, and others have also indicated the concurrence of retained corpus luteum and retention of débris in the uterus, such as occurs in pyometra, hydrometra, foetal mummification, and maceration. As Williams rightly points out, these corpora lutea are usually central or embedded. They do not protrude above the surface of the ovary. Nielsen maintains that the corpora lutea found in cases of pyometra are the result of ovulation subsequent to parturition and not to persistence of the corpus luteum graviditatis. He believes that there is no question of persistence of the corpus in cases of pyometra.

There is every possibility of persistence accompanying endometritis, which cannot be diagnosed clinically in some cases. It is unlikely that such corpora lutea remain for months or indefinitely unchanged histologically. They usually undergo cystic degeneration, or become reduced in size, and sclerotic.

The metabolic changes in the organism which are associated with functional sterility may produce a degree of persistence or at least a delay in regression of the corpus luteum. As previously stated, the great cause of functional sterility in South Africa is obesity due to improper feeding and insufficient exercise in the beef-breeds and feeding for high milk records in the milk-breeds. Richter has noticed persistence in cattle kept constantly in the stall during winter and early spring. In many such animals the interovulation periods are long and irregular, varying from weeks to months. The corpus luteum which develops after ovulation may show little change in size at a clinical examination for several weeks. It, however, slowly decreases in size and sinks to the level of the surface of the surrounding ovary surface. It is no longer easily palpable per rectum. The ovaries are small and there is no evidence of ripening Graafian follicles. However, at a post-mortem examination section of the ovary usually reveals a corpus luteum of considerable size, and brownish or brownish-grey in colour. There is apparently sufficient hormone secretion to influence the development of further Graafian follicles.

The ovaries of cattle suffering from functional sterility because of obesity are as a rule smaller and weigh less than normally fertile cattle of the same age and breed. Histologically the ovaries of these cattle are normal in that they contain no pathological recognizable lesion. There are, however, no large follicles present such as one would expect to find in the opposite ovary to that containing a corpus luteum in a regressive stage.

The corpus luteum in the early stages, that is, for six or seven weeks after ovulation, is histologically unchanged, but as regression occurs, it becomes fibrous and there is a gradual decrease in the lutein cells.

It is difficult to say what influences the duration of regression, as shown by the duration of the interovulation period in the same animal. One would expect that the length of the interovulation periods in the same animal, without visible change in condition, would be approximately equal. This, however, is not the case. Regression so as to allow ovulation takes place in some cases in a few weeks, in others it extends over months.

Low condition has a somewhat similar effect on the ovaries. Attention has already been drawn to the absence of oestrum during periods of drought in South Africa. Such dry periods have a sequel in that the shortage of grass and food in general produces a condition of malnutrition. When the condition reaches a low standard, subfunction of the ovaries occurs. This static state continues until after the first rains, when with the increase in condition the normal physiological activity of the ovaries is again evident. The corpus undergoes normal resorption and ovulation occurs at normal intervals.

Diagnosis.

Persistence of the corpus luteum or delayed regression is associated with absence of oestrum or abnormally long interovulation periods. Wester maintains that the corpus luteum which is not resorbed does not prevent the development of a new follicle. He says one frequently sees follicles ripen and ovulation takes place although the ovary still contains a fairly large corpus luteum. He further states that the oestrum following expression of the corpus luteum is not associated with ovulation. The statement of Wester is not in agreement with the observations of other authorities. Williams, for instance, has seen conception follow the service during the oestral period following expression. Similar observations have been made here when the corpus has been expressed from an apparently normal animal.

The diagnosis of persistence or delayed regression of the corpus luteum can be made only by frequent clinical examinations of the ovaries, so that the possibility of silent heat, as described by Nielsen, Strodthoff, Küpfer, Hammond, Richter, Frei, and others, may not be overlooked. Clinical examinations made at weekly intervals will enable a determination to be made of any changes which occur in size or consistency of the corpus. It is on the results of these frequent examinations that the statement regarding the rarity of persistence in normal cattle is made. Had the diagnosis been made on one clinical examination, which showed the presence of a corpus luteum, followed by absence of oestrous symptoms, the percentage of retention recorded would have been higher. Hess maintained that a persistent corpus luteum and a normal corpus could be differentiated at a clinical examination by the fact that the former is smaller, firmer, and less prominent. Opperman, Stalfors, Richter, and Frei point out the extreme difficulty in differentiation at a clinical or post-mortem examination.

Since persistence or delayed regression of the corpus is considered secondary, one must expect to find pathological lesions of the genitalia or conditions associated with functional sterility concurrent.

In many cases where the corpus luteum has become embedded it will not protrude above the surrounding ovarian surface. It then becomes exceedingly difficult to make a definite diagnosis. The ovary may be somewhat large, round, or ovoid, but the corpus luteum if deeply embedded cannot be felt. However, the concurrent symptoms in these cases usually justify the diagnosis of persistence or delay in regression.

Prognosis.

In view of our belief that the condition is secondary, the prognosis appears to depend entirely on the primary pathological lesion. It is good in cases of subacute metritis which has been treated early. When it occurs concurrently with pyometra, hydrometra, or old-standing cases of metritis chronica catarrhalis, it is exceedingly doubtful. If tubal disease is present and diagnosable, it is hopeless as a rule.

When delay in regression is a concurrent symptom with metabolic change in the organism, associated with sterility, the prognosis can be considered good unless the case is one of old-standing functional sterility, in which histological examination as a rule shows atrophy of the uterine mucosa. These old-standing cases have been found, with one or two exceptions, most obstinate in treatment, while functional sterility in young cattle responds readily to treatment.

Treatment.

Treatment is first based on overcoming of the primary pathological lesion. However, when there is no clinical evidence of disease of the genitalia, it is considered advisable to look upon the possibility of such a condition as very real. All cases are treated by uterine irrigation and massage, as indicated under diseases of the uterus. If the corpus still persists and shows no evidence ot regression within three weeks after the commencement of treatment, it is expressed by pressure through the vagina. This combined method of treatment has been recommended by Albrechtsen, but the expression of the corpus has been delayed until the uterus has had four to six irrigations.

In the case of embedded corpus, expression is sometimes an exceedingly difficult operation requiring considerable pressure. This difficulty has been pointed out by Williams, Richter, and others. In cases of pyometra in which the ovary cannot be reached from the vagina until the contents of the uterus are removed, the contraction of the uterus, which takes place within two to three weeks of treatment, allows of the usual expression by vaginal pressure.

Williams and Opperman have advised the incision of the ovary with the cyst knife, when compression fails. Williams has successfully expressed corpora lutea in this manner, by first incising and then applying pressure through the vagina. He suggests laparotomy to allow of direct ovarian manipulation when vaginal or rectal pressure fails to dislodge a retained corpus.

Several observers have recorded success in the treatment of persistent corpus luteum by expression alone. It is quite probable that expression would favourably influence a slight catarrhal metritis by producing contraction, hyperaemia, and allowing the development of Graafian follicles, since oestrous symptoms usually appear on the third or fourth day following. The good results in these cases of catarrhal metritis, which are clinically diagnosable, following simple expression, may possibly account for an incorrect diagnosis that the condition is a primary phenomenon.

Delay in regression accompanying metabolic disturbance must be treated according to the requirements of individual cases. Obese cows and heifers have been put on to a weight-reducing ration, with sufficient exercise. The genitalia are stimulated to physiological activity by bi-weekly massage. They are put on to a balanced ration with a liberal allowance of green food and bonemeal. The corpus luteum is not disloged. Young cattle quickly react to this form of treatment. The rhythm of the oestrous cycle is usually quickly restored.

Various other methods of treatment are being tried in old-standing obstinate cases. The work is as yet incomplete, but the most successful results have been obtained from a combination of the above method with bi-weekly intravenous injections of whole ovary extract and ovarian grafting.

Cows and heifers, in which low condition can explain the subfunction of the ovary, quickly react to a suitable balanced ration with green food in abundance and ovarian and uterine stimulation by massage. In these cases the use of aphrodisiacs, as recommended by Oppermann, Nielsen, and others, have been found of little or no value in arousing the ovaries to activity.

Similar treatment can be used in heavy-milking cows in which subfunction of the ovaries is evident. Uterine massage, whole ovary extract, a liberal ration of green food, with suitable exercise and company with the bull, as a rule, overcome the difficulty within four to six weeks.

(VI) *Induration.*

Induration of the ovary has been observed in a small percentage of the cows slaughtered because of incurable sterility during these investigations. Sclerosis of the ovary in the cow has been described by several authorities on disease of the reproductive organs in cattle, including Frei, Hammond, and Zschokke.

It appears that the condition is secondary to primary disease of the uterus or the tubes or to old age. The cases which have come to notice in younger cattle have been associated with perioophoritis, salpingitis, and pavilionitis, with encapsulation of the ovary within the ovarian pocket or the fimbriated extremity of the tube. The association between primary inflammatory change in the uterus and the tubes has been described by Nielsen, Bertschy, Zschokke, Posselt, and others. The possibility of early inflammatory change in the ovary resulting from infection followed by organization of the cellular elements appears likely. Zschokke, however, suggests possible toxic influence as a stimulant to the growth of fibrous tissue, the toxins responsible being developed in metritis. Hammond mentions that frequent ovulation, followed by corpora lutea and corpora albicantia, is the cause of induration in old cows.

In the cases encountered here, the ovary has been difficult to palpate, as it was surrounded by fibrous adhesions, which passed between it, the mesosalpinx, the broad ligament, and the uterine horn. (Figs. 29, 43, Appendix II.) It has been seen as a unilateral and a bilateral condition. When bilateral, oestrum and ovulation have been in abeyance. When it was unilateral, oestrum has occurred, but at irregular intervals. The irregularity of oestrum in all pathological conditions of the ovary has already been discussed.

On post-mortem examination the ovary was encapsuled by connective tissue adhesions, which were stripped off only with difficulty. The surface was rough and greyish-white in colour. The ovary was smaller than normal or not increased in size. It was firm on palpation. There was no trace of Graafian follicle or corpus luteum apparent through the surface.

On section, the parenchyma was firm, and greyish-white in colour. Traces of lutein tissue were sometimes seen. No macroscopic follicles were present. Small central cyst-like structures have been observed.

The hypertrophied sclerotic ovary described by Frei and others has not been seen.

Histological examination shows the absence of germinal epithelium. The parenchyma is represented by more or less dense connective tissue. (Fig. 86, Appendix III.) No follicles may be apparent, or here and there follicles undergoing atretic degeneration may be present. Corpora albicantia have been seen. Tertiary follicles were never encountered. Small cysts with or without epithelial lining may be present. Frei describes the presence of small follicles and multiple or single cystic degeneration. He has seen sclerosis in the sheep in which he describes the absence of primary follicles, corpora lutea, and germinal epithelium. The stroma showed much young connective tissue and towards the surface a round-cell infiltration.

Sclerosis of the ovary as a primary disease plays no important rôle as a cause of sterility in cattle, since it is apparently a sequel to diseases of the uterus and the tubes, which in most cases gravely endanger the fertility of the animal. When established, it is unlikely that any form of treatment will again produce physiological activity. The pathological change seen in our cases has been irreparable. Treatment must be confined to prevention by applying suitable therapeutic measures in diseases of the uterus and the fallopian tubes. Further, care must be taken in manipulation of the ovary when treating cysts or retained corpora lutea. Manipulations must be as gentle as possible. Rough careless handling, which causes severe crushing, is contra-indicated, as oophoritis may follow. It is quite possible that the resulting inflammatory phenomena may resnlt in organization of the cellular elements with sclerosis and atrophy of the normal ovary parenchyma.

FUNCTIONAL STERILITY.

Functional Sterility, the cause of which appears to be due to metabolic disturbance of the organism, has been referred to frequently throughout this paper. The general causation has been described in the introduction. The ovarian change and its influence on the oestrous cycle has been discussed in the chapter "Diseases of the Ovary." The diagnosis, methods of treatment, and the results obtained by treatment, have also been briefly referred to.

The subject of functional sterility is at present receiving attention at this Institution. However, the number of animals which has been under observation is considered insufficient. It is, therefore, intended to postpone writing up the resnlts of our observations until a later date when they will be more conclusive.

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APPENDIX I.

In this Appendix details are given concerning the general appearance, history, gross examination, post-mortem examination, histological examination of the genitalia and the endocrine glands, treatment, and the cause of sterility in the case of cows slaughtered as incurable during these investigations.

CASE I. (See Appendix II, Fig. 1.)

AYRSHIRE COW O.2, 5 YEARS OLD.

Date of first examination, 17.8.25. *Date of slaughter*, 17.8.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition is poor in the extreme. Has lactated. There is a peculiar foetid smell from the animal. The tail and hind-quarters are stained with a yellowish thin purulent material. No. of calves—1. The afterbirth was retained and removed by the owner. Since parturition there had been a purulent discharge from the vulva. The cow had not shown oestrum since parturition.	The mucosa of the vulva is very pale and slightly icteric. There is a thin purulent foetid discharge which issues in small quantities. The vagina is also pale and there is a small quantity of pus on its floor. The cervix appears closed. Its position is represented by a cicatrix. The uterus is greatly distended and tense. Both horns are equal in size. Its wall appears thin. It is fluctuating. There is no evidence of cotyledons. The right ovary contains a corpus luteum. It is about the size of a small hen egg. The left ovary is similar in size and normal in consistency. The fallopian tubes cannot be palpated. Broad ligaments are elongated and tense. *Diagnosis;* Uterine abscess, stenosis of the cervix.	Vulvar and vaginal mucosa are pale-yellowish in colour and somewhat granular in appearance. There is a small amount of foetid purulent material present in the vagina. There is no sign of cervix uteri. Its place is taken by a cicatrix, 1½ inches in diameter and 1 inch in thickness. No opening can be found connecting the vagina with the uterus. The pus in the vagina must have come from the mucosa. The uterus is enormously distended and tense, as are the uterine horns. On opening the uterus it contained 21 litres of very thin liquid pus, which is extremely foetid. The uterine walls are thin and flaccid. The mucosa is yellowish-grey in colour. There is no macroscopic evidence of cotyledons. Both fallopian tubes are enlarged, somewhat tortuous, and firm. The broad ligaments are stretched.	'Right ovary, 5 × 3½ × 3½ cm., shows a smooth surface except for the presence of a corpus luteum, which is very prominent. On section, it contained a corpus 2·5 cm. in diameter, which extends 1·5 cm. into the ovary; its colour is orange yellow. There are several atrophied corpora lutea and some small follicles. Left Ovary, 4½ × 2½ × 2½ cm., contains many atrophied corpora lutea and a ripening Graafian follicle. The ovary tissue is greyish-white and firm in consistency.	This cow was destroyed during an examination for sterility in the field. The genital organs only were collected for examination. *Vagina*—catarrhal vaginitis. *Cervix*—cicatrical tissue only seen. *Uterus*—pyometra. *Fallopian tubes*—salpingitis. *Ovaries*—No change. The large retained corpus luteum of pyometra showed normal structure.	None. Case hopeless.	Uterine abscess.

CASE 2. (See Appendix II, Fig. 2.)

AYRSHIRE COW 0.37, 17 YEARS OLD.

Date of first examination, 20.8.25. *Date of slaughter*, 16.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
This cow was one of the best Ayrshires imported into South Africa. She had won many championships. Now she shows old age to a marked degree. Her condition is poor, but the cow is very active. She feeds well. The udder is dry No. of calves, several. Date of last parturition, 1924. The cow has not shown oestrum since last parturition. Contagious abortion test not available. Tuberculin Test not available.	Vulva is pale in colour. There is a slight yellowish flocculent discharge. The vaginal mucosa is normal, but there are traces of yellowish purulent material. The os uterus admits the finger for a distance of 3 cm. The cervix cannot be withdrawn for examination. Per rectum the cervix is not easily palpable, but it appears to be somewhat short and increased in circumference. The uterus is enlarged to the size of a small football. It is firm and non-compressible. The uterine horns are not involved in the tumour formation. They are not easy to palpate. They are flaccid and project from the antero-inferior aspect of the tumour. The left horn is shorter than the right. The fallopian tubes are normal in size and consistency. Right ovary is round, slightly fluctuating in areas, and about the size of a small hen egg. Left ovary is closely adherent to the tumour. It is oval in shape and firm in consistency and about the size of a small hen egg. Broad ligaments are elongated. *Diagnosis*; New growth on uterus.	The specimen was sent by the owner. The vulva and vagina are not available for examination. The cervix is ragged and pale in colour. Its length is 4 cm. The anterior extremity is dilated and funnel-shaped so that it runs smoothly into the uterine body. The os uterus is patent. The transverse folds are not distinct. The uterine body, 28 × 20 × 18 cm., is enlarged and firm. The serosa is smooth, glistening, and pinkish-yellow in colour. On section the wall is thin, varying in thickness from ·3 to ·5 cm. The inner surface is smooth and dirty grey in colour. It is easily detached from the tumour, which it encapsules, except one area about 5 cm. in diameter, on the floor, which is firmly adherent to the tumour. The enlarged saċ is filled with an ovoid new-growth weighing 2½ kg. The surface of the tumour is irregular, and greyish-yellow to greyish-green in colour. It, for the most part, lies free in the enlarged uterine sac. On the ventral surface it is attached by a well-marked neck, about 5 cm. in diameter, to the left side of the wall of the uterus. At the area of attachment the wall of the uterus is 4 cm. in thickness. On section, the tumour presents two zones. The peripheral zone is from 1·5 to 7 cm. in diameter. It is grey-greenish-yellow in colour and brittle. The medulla is pale whitish-pink and somewhat firm. It sends blunt processes into the cortical zone. At the junction of these two zones there is an irregular dark reddish demarcation. Right horn, 16 cm. It is flaccid and contains a small amount of opaque exudate. The mucosa is greyish. There is no evidence of normal cotyledons. Left horn, 12 cm. It is similar in appearance to the right on section. Both fallopian tubes appear normal in size and consistency. The broad ligaments are elongated.	Right ovary, 4 × 3 × 3 cm., is roundly oval in outline with a smooth serous covering. It is fluctuating on palpation. On section, it contains five Graafian follicles. The follicles contain a clear slightly viscid fluid and have smooth walls. The remainder of the ovarian tissue is firm and pale greyish-white in colour. Left ovary, 4 × 2·5 × 2·3 cm., is oval in outline and very firm in consistency. It is closely adherent to the swelling, between which and the ovary there are firm fibrous attachments. On section, it shows several corpora albicantia. The tissue is pale greyish-white in colour and very firm. There is no evidence of Graafian follicles or recent functional activity.	*Cervix*—induration. *Horns*, uterus—metritis chronica catarrhalis cystica. *Body*—metritis chronica purulenta with atrophy of the muscularis. *Fallopian tubes*—normal. *Ovaries*—Left: follicular atresia, induration, fibrous filaments, small area of calcification apparently of follicular origin. Right: normal follicles, several corpora albicantia. *Tumour*—mixed sarcoma; the cortex is undergoing fatty degeneration and necrosis.	No treatment was tried. The cow was not kept under observation between the date of examination and the date of slaughter, as the owner refused to part with her for sentimental reasons.	Mixed sarcoma in pars indivisa of the uterus, concurrent with metritis chronica purulenta. The mucosa of the horns showed metritis chronica catarrhalis with commencing cystic formation.

CASE 3. (See Appendix II, Fig. 3.)

HEREFORD COW No. T.U.C. 1, 7 YEARS OLD,

Date of first examination, 14.2.27. *Date of salughter*, 24.2.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition fair. Weight 1,270 lb. The animal has the typical appearance of a nymphomaniac. The head is coarse. The pelvic ligaments are relaxed. The vulva is large. The vulvar lips are soft and flabby. The oestral periods are at short interovulation intervals. No. of calves, 2. Date of last parturition, May, 1924. Contagious abortion test, negative. Tuberculin test, negative.	The vulvar lips are enlarged and flabby. The vagina is large. It contains a quantity of somewhat opaque mucus. The cervix has a slightly ectropic 1st fold, mucosa pale yellowish-pink. The lumen contains some tenacious mucus. The circumference of the cervix is normal. The uterus is mostly abdominal. It is soft, thin-walled, and flabby. It does not react readily on palpation. The horns are equal in size, soft, and flabby. Both fallopian tubes are enlarged at the uterine attachment. The enlargement decreases gradually towards the ampulla. They are firm on palpation. Left ovary is enlarged and cystic. The right ovary is normal in size and consistency. Broad ligaments appear to be unchanged. *Diagnosis*: Metritis chronica catarrhalis, salpingitis, cystic ovaritis.	Vulva normal. Vagina contains a quantity of opaque tenacious mucus; mucosa pale yellowish-pink. Cervix 10 × 7 cm.; width of wall 3·5 cm. The os uterus contains a small amount of tenacious mucus. Uterus—body, 11 × 3·5 cm.; width of wall, 1 cm.; left horn, 22 × 3 cm.; width of wall, ·5 to 1·2 cm.; right horn, 22 × 3 cm.; width of wall, 1 to ·5 cm. Cavum uteri contains a small quantity of opaque mucus. Mucosa is yellowish-grey in colour. It is smooth and moist. There are some pin-point red spots. Scattered throughout the mucosa are several small cyst-like elevations varying in diameter from 1 mm. to 1 cm. They are scattered with equal frequency over the pars indivisa and the horns. They do not extend into the zona muscularis, but are confined to the mucosa. The cotyledons are degenerated. There are continuous ridges of elevated mucosa, on the summit of which these cyst-like structures also appear. The serosa of the uterus is smooth, and yellowish-pink in colour. Both fallopian tubes are enlarged. The enlargement is firm and gradually disappears towards the ampulla. The right tube shows a local convolution about its middle. Here there are fibrous adhesions on the mesosalpinx. The fimbriated extremity of both tubes appears unchanged. The broad ligaments are not changed.	Left ovary, 5 × 4 × 3·2 cm. It is fluctuating throughout. The capsule is thin and transparent in places so that the liquid cystic contents can be seen. On section, is almost entirely cystic. There is one large cyst and two small cysts. These are separated by thin fibrous septa. They contain a clear slightly viscid fluid. The ovarian tissue which remains appears lamellated and moist. It is whitish-pink in colour. The right ovary, 4 × 3 × 2 cm., is firm in consistency and smooth on its surface. Two cyst-like areas are present towards its anterior pole. The remainder of the capsule is pale in colour. On section the ovary is almost entirely composed of firm greyish to whitish-pink tissue. There are two small cyst-like structures, apparently normal. Graafian follicles present.	*Epiphysis*.—Normal. *Hypophysis*.—Normal. *Thyroid gland*.—Normal. *Adrenal gland*.—Hyaline degeneration of the cortex, especially the zona fasiculata and zona reticularis. *Thymus gland*.—Normal. *Pancreas*.—Normal. *Supramammary lymph gland*.—Normal. *Mammary gland*.—Normal. *Cervix*.—Normal. *Uterus*.—Metritis chronica catarrhalis cystica. *Fallopian tubes*.—Fibrous of the villi with atrophy of the epithelium. *Ovary*.— Left: Cystic ovaritis, Graafian follicle cysts. Right: Small cysts, Graafian follicle cysts.	Condition considered hopeless. No treatment tried.	Metritis chronica catarrhalis cystica. Salpingitis. Cystic oophoritis (Graafian follicle cysts).

CASE 4. (See Appendix II, Fig. 4.)

FRIESLAND COW 1212, BORN 1909.

Date of first examination, 24.7.25. *Date of slaughter*, 3.12.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,550 lb. There is an appearance of senility. The udder is non-lactating, very large, and pliable. Number of calves, 6. Last parturition, 3/9/19. While under observation the animal had three normal oestral periods. Contagious abortion test, negative. Tuberculin test, negative.	Vulva shows nothing unusual. There is a small quantity of thick opaque mucus present in the vagina. The cervix is very ragged; the first cervical fold is ectropic. It is broken at irregular intervals by deep fissures. It is firm on palpation. The ostium uterinum externum admits the little finger to its entire depth. The circumference of the cervix is larger than normal. The uterus appears normal in size but lacking in tone. The fallopian tubes are not changed. Right ovary, size of small hen egg. Left ovary, lobulated and fluctuating. On slight pressure it collapses, leaving a small firm ragged structure. Broad ligaments are normal. *Diagnosis;* Senility, atony of the uterus.	Vulva and vagina show nothing unusual. The cervix is firm and the cervical wall is thickened. It is almost cartilaginous in texture. The transverse folds are very prominent. The third cervical fold is extremely firm and cartilaginous on section. Uterus body, 10 cm.; right horn, 15 cm.; left horn, 13 cm. The uterus and uterine horns show a greyish-red mucosa. The cotyledons have degenerated. The right horn is larger than the left. The fallopian tubes are unchanged in size. The broad ligaments appear unchanged.	Right ovary, 4 × 2½ × 2 cm. On its lateral surface there is a cyst, 1¼ cm. in diameter, which contains a clear fluid. The cyst appears to be attached to the surface of the ovary. On section, the ovary shows a corpus luteum about 2 cm. in diameter. There are several atrophied corpora lutea present. Several small follicles are visible on the surface of section. The ovarian tissue is greyish-white and firm in consistency. Left ovary, 3 2 1½ cm., shows a similar cyst on its lateral surface, about 1¼ cm. in diameter. On section, the ovary shows the presence of several atrophied corpora lutea. Several small follicles are visible on the surface. The ovarian tissue is whitish grey in colour and firm in consistency.	*Epiphysis.*—Corpora amylacea numerous. Local increase in fibrous tissue, which shows a few areas of commencing calcification. *Hypophysis.*—Normal. *Thyroid.*—Normal. *Adrenals.*— (1) Normal; (2) Haemorrhage into the cortex. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Atrophy of the mucosa and commencing cystic formation in the glands. *Fallopian Tubes.*—Normal. *Ovaries.*— Left, Sclerosis. Right, Normal corpus luteum.	No treatment tried.	Senility with degeneration of the uterine mucosa.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis	Weight	0·6 gm.
Adrenals—Right, 5 × 5 × 2 cm.	,,	20·0 gms.
Left, 7·5 × 4 × 1·5 cm.	,,	19·0 ,,
Thyroid—Right half, 4·5 × 7 × 1 cm.		
Left half, 5 × 6·5 × 1 cm.		
Length, including isthmus, 18·5 cm.		

CASE 5. (See Appendix II, Fig. 5.)

FRIESLAND COW 1213. BORN 13.5.13.

Date of first examination, 24.7.25. *Date of slaughter*, 13.4.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1.464 lb. There is very slight relaxation the pelvic ligaments. The appearance is otherwise normal. The udder contains small quantity of opaque watery fluid mixed with yellow flocculi. The animal had suffered from acute contagious granular vaginitis in September and October, 1922. No. of calves, 6. Last parturition 8/2/'22. Since the beginning of 1922 periods of oestrum have been irregular and the cow had been bred several times without success. Contagious abortion test, negative. Tuberculin test, positive.	The vulva shows a small recent wound close to the lower commissure. There are some dormant nodules of chronic granular vaginitis. The vagina contains a small quantity of opaque tenacious mucus mixed with yellow flocculi. The cervix is ragged and the first cervical fold is ectropic. Its mucosa is red in colour. The os uterus admits one finger easily to the third cervical fold. Per rectum the cervix appears abnormally firm and somewhat elongated. The uterus is enlarged, mostly abdominal. There is irregularity in the size of the horns, the left being larger. The uterus is flaccid and lacking in tone. Both fallopian tubes are normal in size. The right ovary is the size of a small hen egg, and firm: it contains a projecting corpus luteum about 1·5 to 2 cm. in diameter. The left ovary is the size of a walnut, firm in consistency. No evidence of developing Graafian follicles can be felt. The broad ligaments are normal. *Diagnosis:* cervicitis, metritis chronica catarrhalis, corpus luteum persistens. During treatment this cow developed cystic oophoritis and hydrosalpinx.	The vulva shows a few nodules, greyish-yellow in colour. The vagina contains a small quantity of thick tenacious mucus. The mucosa of the vulva and vagina is yellowish-pink in colour. Cervix, 11 3·2 cm.: mucosa is pale. The first cervical fold protrudes through the ostium uterinum externum. it is ragged. The os admits one finger easily. On section it cuts almost as hard as cartilage. Uterus body, 13 8 cm.; left horn, 32 cm.; right, 27 cm.; width of wall, 1·5 cm. The peritoneal surface of the uterus shows fibrous adhesions. The wall is soft, moist, and flaccid. The mucosa is greyish-pink to greyish-white in colour; it is partly transparent and very moist. The cotyledons are represented by greyish-yellow non-elevated patches. Both fallopian tubes are markedly enlarged, convoluted, and fluctuating; their greatest diameter is 2 cc. immediately behind the pavilion. They contain a clear watery fluid. In some parts they are firm and occluded. Both fimbriated extremities are adherent to the ovary; they are enormously enlarged and fluctuating. They contain a clear fluid. The broad ligaments appear somewhat elongated.	The right ovary is rounded, diameter 5 cc., covered entirely with fibrous adhesions. It is adherent to the fimbriated extremity of its tube. It is entirely cystic and fluctuating. On section, it contains a single large cyst 3·5 cm. in diameter, which is filled with clear yellowish slightly viscid fluid. It is surrounded by a pale yellow capsule, 1mm. in thickness, outside which is a greyish-white capsule up to ·5 cm. in thickness. Scattered over this latter capsule are the fibrous filaments above described. The left ovary is soft to the feel, 4 3 2·5 cm.; it also is covered with fibrous adhesions which attach it to the uterus on one side and the fallopian tube on the other. On section, it contains four peripheral cyst-like structures varying in size from ·5 to 1·5 cm. in diameter. These contain a clear yellowish, slightly viscid fluid. The inner surface of the cyst wall is pale and smooth. The remainder of the ovary is pale greyish-white and fibrous looking. It contains many large blood-vessels. There is no evidence of a recent corpus luteum, as one would expect in a cow which had recently shown oestrum.	*Adrenal.*—Right and left, normal. *Epiphysis.*—Corpora amylacea very numerous. *Hypophysis.*—Vesicle containing colloid in pars distalis. *Thyroid gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Fibrosis. *Supramammary lymph gland.*—Fibrosis. *Mammary gland.*—Numerous concretions. *Uterus.*—Perimetritis, metritis chronica catarrhalis, and early stages of metritis tuberculosa. *Fallopian tubes.*—Salpingitis tuberculosa — tubercular lesions older than in the uterus. *Ovary.*— Right, Graafian follicle cyst, fibrous adhesions. Left, small cysts and several atretic follicles. Oedema.	24/7/25: Corpus luteum expressed. From this date until the 15/1/26 the cow received 14 treatments to the cervix by swabbing with Lugol's iodine, five uterine irrigations with 1 per cent. Lugol's iodine. At each treatment the uterus was massaged.	Metritis chronica catarrhalis, cervicitis, retained corpus luteum. This was followed by salpingitis and cystic oophoritis; the tubercular invasion of the fallopian tubes and the uterus apparently occured while the cow was under observation.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·3 × ·7 × ·6 cm.	Weight 0·3 gm.	Thyroid—Right half, 4·8 × 9·8 × 1 cm.
Hypophysis, 2·7 × 2·8 × 2·8 cm. . . .	„ 4·02 „	Left half, 4·8 × 7 × ·9 cm.
Adrenals—Right, 8·2 × 4 × 1 cm. . .	„ 21·0 „	Length, including isthmus, 19·5 cm., weight 47 gm.
Left, 5 × 5 × 1·5 cm.	„ 19·0 „	

CASE 6. (See Appendix II, Fig. 6.)

FRIESLAND COW 1215, AGE 13 YEARS.

Date of first examination, 24.7.25. *Date of slaughter*, 25.2.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,530 lb. Normal in appearance. Udder has lactated, but is now dry. Udder tissue normal. No. of calves—4: First 4.6.17 last 3.10.20. The cow suffered from acute contagious granular vaginitis September, 1922. Oestral periods irregular, occurring at intervals of 4–6 weeks. Occasionally a period of 2–3 months occurred without an oestral period appearing. Contagious abortion test—negative. Tuberculin test—negative.	Vulva, mucosa normal. Vagina small quantity of clear tenacious mucus, mucosa normal. Cervix—1st cervical fold ragged, ectrophic, and firm. The os admits one finger to its entire depth. The circumference of the cervix is enlarged and consistency firm. Uterus mostly abdominal; it appears thick-walled and firm. Reacts quickly by contracting on palpation. Both fallopian tubes normal. Both ovaries normal in size, firm in consistency. Broad ligaments appear normal. *Diagnosis*: metritis chronica catarrhalis, with some fibrosis of the uterine wall, cervicitis chronica with fibrosis of the cervical wall.	Vulva and vagina normal. Mucosa pale-yellowish in colour. Cervix, 10 × 8 cm., width of wall, 4 cm. Mucosa pale-yellowish in colour. The os admits the little finger in its posterior portion. First cervical fold ectropic. First and second cervical folds hypertrophied. There is a small amount of tough mucus in the lumen. Uterus-body, 10 × 4·5 cm.; width of wall, 1·5 cm.; left horn, 16 × 2·5 c.m.; width of wall, 1·2–·7 c.m.; right horn, 18 × 3·5 cm.; width of wall, 1·5–·5 c.m.; Serosa smooth, yellowish-pink in colour. On Section, uterus contains a small quantity of opaque mucus. Mucosa dark greyish-pink in colour. Moist. Cotyledons, especially in the right horn, are prominent yellowish-white in colour. They vary in diameter up to 2 c.m. Both fallopian tubes are normal. The right suprarenal gland shows a lesion 2 × 1 × 1 c.m. which is surrounded by a tough white capsule. It is embedded in the cortex and medulla. On section, it shows small cavities separated by fibrous septa. Some of the cavities contain a dark red material of the consistency of coagulated blood, others show an amber-coloured jelly-like material.	Left ovary, 4 × 3·5 × 2·5 c.m. Surface shows numerous small cyst-like bodies, apparently normal follicles. The corpus luteum of the last interovulation period is present. Traces of old corpora lutea can also be seen on the surface. On section, there is a large corpus luteum 2 c.m. in diameter, orange-yellow in colour and somewhat soft in consistency. There are some normal Graafian follicles. The ovarian tissue is pinkish-white and firm. Right ovary, 4·5 × 2·8 × 2·5 c.m., shows a similar outward appearance to the left. There is one large cyst-like prominence towards its anterior pole, apparently a rapidly developing follicle. On section, shows several apparently normal follicles. There are traces of corpora lutea of former interovulation periods. The ovarian tissue is pinkish-white and firm in consistency.	*Thymus.*—Normal. *Thyroid.*—Normal. *Epiphysis.*—corpora amylacea. *Hypophysis.*—Normal. *Pancreas.*—Normal. *Adrenals*— Left: Medulla rich in blood-vessels, connective tissue and nerves. Right: portions similar to the left. Lesion described appears to be an old haemorrhage which is becoming organized. *Supramammary lymph gland.*—Some fibrosis. *Mammary gland.*—Normal. *Vagina.*—Mucosa infiltracted with neutrophiles. *Cervix.*—Fibrosis. *Fallopian tubes.*—Some slight fibrosis of villi. *Uterus.*—Some enlarged and cystic mucous glands, oedema of the mucosa. *Ovaries.*—Atretic follicles, small cysts lined with epithelium. Oedema.	This cow underwent a course of treatment with irrigation of Lugol's iodine, 1 per cent. in saline, and swabbing of the cervix with pure Lugol's iodine. Uterine massage was also tried at the same time. Although the genital tract appeared normal on rectal palpation after treatment, several services were without result. It was decided to destroy the animal for examination.	Fibrous cervicitis. Some degree of chronic metritis. Microscopic changes very slight. They consist of oedema and some enlarged mucous glands which are local. Most of the mucous glands are normal. Tubes show slight fibrosis of the villous folds. The ovary shows oedema, and some small cysts, lined with epithelium. The microscopic changes are so slight that it is doubtful if the lesione in the genital tract can be associated with sterility.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·6 × 1·8 × 1·6 cm....	Weight 0·5 gm.	Thyroid—Left, 6 × 4·2 × 1·2 cm.
Epiphysis, 1·7 × ·7 × ·7..........	,, 0·4 ,,	Right, 6 × 4 × 1 cm.
Suprarenal—Left, 5 × 4·5 × 1 cm...	,, 11·0 gms.	Length, including isthmus, 24 cm., weight 29 gms.
Right, 6·5 × 3·8 × 1·6.	,, 17·0 ,,	

CASE 7. (See Appendix II, Fig 7.)

SHORTHORN COW 1218, AGE 10½ YEARS.

Date of first examination, 24.7.25. *Date of slaughter*, 8.7.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition very good. Weight 1,460 lb. Appearance normal. The udder does not appear to have lactated: Tissue soft and elastic. There is no history of pregnancy. Contagious abortion test—positive. Tuberculin test—negative. While under observation for 2 years at his Institute the cow showed irregular oestral periods. with very long interovulation periods up to 6.4.26, after which date she developed abnormal sexual desire and would allow copulation at intervals of a week to two weeks, except for four months from 25.6.26 to 5.10.26, when no oestrum appeared. She did not assume the outward appearance of a nymphomaniac.	Vulva does not admit the hand: Mucosa is normal, there is a slight amount of clear mucus present. The vagina from the rectum appears normal. Cervix normal in size and consistency. Uterus small in size, slightly flaccid: Uterine horns small in size and flaccid. Fallopian tubes normal. Left ovary normal: ovary is small. bean-shaped. the crater of a ruptured Graafian follicle can be felt. Right ovary is rounded, normal in consistency and size. Board ligaments normal. *Diagnosis:* functional sterility While under observation and treatment the ovaries became cystic, this was followed by degeneration of the uterine wall with lack of tone and the accumulation of small quantities of fluid.	Vulva: mucosa normal. Vagina: mucosa normal, small quantity of clear tenacious mucus present. Cervix, 8×3 c.m.; width of wall, 1·5 c.m. First cervical fold partly ectropic. mucosa reddish. There is a small quantity of clear mucus present. Ostium uterinum is patent for a large probe. Uterus—body 8 × 4·5 c.m.; width of wall, ·4 c.m.; horns. 11 × 2·5 c.m., width of wall, ·5 c.m. Serosa smooth somewhat reddish in colour. Body and horns contains 40 c.c. of an opaque milky fluid. The mucosa is greyish-red in colour and smooth There is no trace of cotyledons, but here and there a small cyst the size of a pin's head can be recognized. The fallopian tubes appear normal in size, but there are a few fibrous filaments extending across the fimbriated extremity. Broad ligaments normal.	Left ovary, 4 × 3 2·8 c.m., is entirely cystic, fluctuating throughout, thin-walled. On section, there are four cyst-like structures. varying in size from 1 to 2·3 c.m. They contain a clear slightly yellowish fluid. There is little ovarian tissue remaining in the capsule and the septa. It is greyish-pink in colour and moist. Right ovary, 4×3×2·5 c.m., similar in outward appearance to the left. On section, there are five cyst-like structures varying in size from ·7 to 2 c.m. They contain a clear yellowish fluid. There are traces of lutein tissue in the capsule and the septa. The ovarian tissue is pinkish-grey in colour and moist.	*Epiphysis.*—Normal. *Hypophsis.*—Normal. *Adrenals.*—Local fatty infiltration in the cortex. *Thymus.*—Normal. *Pancreas.*—Normal. *Thyroid.*—Normal. *Parathyroid.*—Normal *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Atrophy of muscular wall, atrophy of mucosa, with increase in size and decrease in the number of uterine glands. There is an active process. Some of the glands are enlarged and filled with round cells and neutrophiles. There is also periglandular round-cell infiltration. Slight hyperaemia. *Fallopian tubes*—Normal. *Ovaries.*—Cystic degeneration of the Graafian follicle. Oedema.	25.7.25—17.9.25: Genital massage. 6.4.26—7.6.26: Monthly injection of pituitary infundibular extr. 1–5 c.c., adrenalined 1c.c. 14.2.27—7.4.27: Bi-weekly injection 10 c.c. whole ovary extract intravenoulsy, followed by 2 lb. of sprouted oats daily, given every alternate week. Owing to progressive degeneration of the uterine wall, the case was condered hopeless and the animal destroyed.	Functional sterility followed by atrophy of the uterine mucosa and muscularis. The active process described at the microscopic examination was possibly of more recent origin as were the cystic changes in the ovaries.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·5 × ·6 × ·6 cm....... Weight 0·7 gm.
Hypophysis, 2 × ·7 × ·6 cm........ ,, 0·4 ,,
Thyroid—Left, 5 × 5 × 1·3 cm.....
Right, 6 × 5 × 1·4 cm.
Length, including isthmus, 21 cm., weight 33 gms.

Adrenal—Left, 6·5 × 3·4 × 1·3 cm., weight 13 gms.
Right, 4·5 × 2·4 × 1·4 cm., weight 10·5 gms.
Parathyroid, ·6 × ·4 × ·3 cm.

CASE 8. (See Appendix II, Fig. 8.)

SHORTHORN COW 1219, 9 YEARS OLD.

Date of first examination, 9.7.25. *Date of slaughter*, 1.7.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,250 lb. Normal in appearance. Udder does not appear to have lactated: tissue elastic. This was an imported Shorthorn heifer which was used as a reservoir animal for redwater and gall-sickness vaccine at this Institute for a period of three years. At the age of five years she was transferred to one of the Schools of Agriculture, where she was kept for a period of twelve months and served several times without success. The heifer never conceived. Contagious abortion test—positive. Tuberculin test—negative. She was kept under observation at this Institute for two years prior to slaughter. During this period she developed abnormal sexual desire and would take the bull at weekly intervals for months; this would be followed by a period of anaphrodisia lasting a couple of months.	Vulva does not admit the hand; mucosa normal; vagina from the rectum appears normal; the cervix is small, short, and firm in consistency; uterus is small, entirely pelvic, and of good tone; both fallopian tubes are normal. The right ovary is the size of a walnut, firm in consistency; there is no evidence of Graafian follicles or corpus luteum. The left ovary is the size of a walnut, firm in consistency; there is a small protrusion from the surface, an atrophying corpus luteum. The broad ligaments are normal. *Diagnosis*: functional sterility as the result of being maintained in an extremely high condition without exercise while acting as a reservoir animal. While under observation the cow developed abnormal sexual desire as explained in the previous column. She developed a cystic ovary on the left side which was broken down and did not recur.	The vulva admits three fingers; mucosa normal; vagina, small quantity of mucus which is clear and tenacious. Cervix, 6 × 3·5 c.m.; width of wall, 1·7 c.m.; mucosa is pale pinkish in colour; the ostium uterinum is small in calibre, its anterior extremity admits a large probe only. Uterus-body, 9 × 3·5 c.m.; width of wall, 6 c.m.; horns 12·5 × 2 c.m.; width of wall, ·6 c.m.; serosa smooth; mucosa dark pinkish-red in colour with some small red spots; there is a small quantity of opaque mucus present, about 20 c.c. The cotyledons are not prominent and yellowish in colour. The uterine wall is narrower than normal, especially the mucous coat. The fallopian tubes are unchanged. The broad ligaments are unchanged.	Right ovary, 4 × 2·8 × 2·6 c.m.; normal in consistency; there are numerous normal follicles apparent on the surface; the corpus luteum of the last interovulation period is present, about 1·5 c.m. diameter. On section, there is a large corpus luteum 1·5 c.m. in diameter. It appears normal. The ovarian tissue is greyish-pink in colour and normal in consistency. Left ovary, 3·8 × 2·8 × 1·8 c.m., normal in consistency; numerous normal follicles apparent on the surface and trace of two atrophied corpora lutea. On section, the corpus luteum of the second last interovulation period is present, ·4 c.m. in diameter. It is brownish-yellow in colour. The ovarian tissue is greyish-pink in colour, and normal in consistency.	*Epipysis*.—Pigment in connection with the vessel walls (Melanin); only present at the periphery of the gland. *Hypophysis*.—Pars distalis-hyperaemia. *Pancreas*.—Normal. *Thymus*.—Normal. *Adrenal glands*.—Normal. *Thyroid*.—Vesicles distended with colloid, epithelium mostly flat. *Lymph gland*.—Normal *Mammary gland*.—Normal. *Vagina*.—Normal. *Cervix*.—Normal. *Fallopian tubes*.—Normal. *Uterus*.—Atrophy of the mucosa. Few cystic glands. Hyperaemia. Glands fewer in number than normal. *Ovaries*.—Normal. corpus luteum. Normal follicles. Atretic follicles. Small cyst with epithelial lining. Some oedema.	24.7.25 to 15.1.26: Balanced ration rich in protein and vitamin E; unsuccessful. 6.4.26 to 16.5.26: Bi-weekly injection 2 c.c. pituitary Infundibular ext. and 1 c.c. adrenalined. At this period nymphomania developed and udder became enlarged; unsuccessful. 17.8.26: Grafted with ovary, which contained some follicles and a corpus luteum three days old, in neck; unsuccessful. 14.2.27 to 7.4.27: Bi-weekly injection of 10 c.c. lutein ext. intravenously; unsuccessful.	Functional sterility of old-standing, followed by degeneration of the uterine mucosa.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·6 × 2 × 1·6 cm.	Weight 3·3 gms.
Epiphysis, 1·5 × ·6 × ·6 cm.	„ 0·5 gm.
Adrenal—Left, 4·6 × 4 × 2 cm.	„ 13·5 gms.
Right, 6·5 × 3·8 × 1·2 cm.	„ 15·5 „

Parathyroid, ·5 × ·4 × ·3 cm
Thyroid—Right half, 8 × 4·5 × 2·2 cm.
Left half, 6 × 4·5 × 1·8 cm.
Length, including isthmus, 20 cm., weight 49·5 gms.

CASE 9. (See Appendix II, Fig. 9.)

NATIVE COW 1293, BORN 1920.

Date of first examination, 21.10.25. *Date of slaughter*, 29.10.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. The head is slightly coarse and masculine in appearance. The animal is otherwise normal: the udder does not appear to have lactated. The animal has never calved. (As the cow ran on the veld with the breeding herd, it would be quite possible, however, that she was served and aborted, the abortion being unnoticed.) Contagious abortion test—negative. Tuberculin test—negative.	Vulvar opening is small, and faces slightly upwards, assuming the position of "horizontal vulva." A few small necrotic areas are present on the mucosa. It does not admit the hand. The vagina cannot be examined internally. The cervix is enlarged in diameter and is firm. The uterus is mostly abdominal. It is enlarged and firm. On pressure it has a grating feel. It is about 4 inches in diameter. Immediately in front of the cervix, the uterine body for a distance of three inches is soft and compressible. The uterine horns are two inches in diameter. They are firm and have a grating feel on pressure. The fallopian tubes are firm and much enlarged, almost to the size of a lead pencil. Left ovary—size of a pigeons' egg. It contains a corpus luteum. Right ovary—smaller in size. It appears normal in consistency. Broad ligaments normal. *Diagnosis*: pyometra.	The mucosa of the vulva and vagina is unchanged, except for a few necrotic patches. The cervix is enlarged and firm. Its mucosa is normal in colour. Uterus-body, 16 × 7 cm.; horns, 23 × 5 cm. The horns and the anterior portion of the body are firm and grating on pressure On section, the mucous membrane is diffusely covered with a yellowish-green granular material, which is adherent to the wall and is also present in the depth of the wall in circumscribed foci in several places. The wall is thickened. It is present from the cornual apices to the cervix, but is not so marked in the caudal portion of the body. The material is drier and more gritty in some places. The fallopian tubes are enlarged and prominent, almost cord-like. They are 3 to 4 mm. in diameter. They are larger at the pavilion and cornual attachment than in the middle of the tube. Broad ligaments are normal.	Right ovary, 3 × 1·3 × 1 cm.; oval in shape and smooth. It contains several small Graafian follicles up to 2 mm. in diameter. There are also present two atrophying corpora lutea. The ovarian tissue is greyish-white and firm. Left ovary, 3 × 2·2 × 1·5 cm.: its surface shows a firm prominence 1·5 cm. in diameter. It is otherwise smooth. On Section, there is present an orange-yellow corpus luteum, 2 cm. in diameter, which extends deeply into the ovarian tissue. There are three small developing Graafian follicles 2 to 4 mm. in diameter. The ovarian tissue is greyish-white and firm in consistency.	*Epiphysis.*—Corpora amylacea, large and very numerous. *Hypophysis.*—Normal. *Adrenals.*—(1) hyperaemia of zona reticularis; (2) normal. *Thyroid.*—Vesicles, mostly large and distended with colloid. *Lymph gland.*—Normal *Vagina.*—Local ulceration of the mucosa. *Cervix.*—Fibrosis. *Uterus.*—Chronic changes of pyometra, associated with calcification in the mucosa and muscularis (tubercular lesions not demonstrable histologically.) *Fallopian tubes.*—Salpingitis. *Ovaries.*—Corpus luteum shows an apparent increase in the connective tissue of the stroma. Normal follicles present.	Prognosis hopeless. Cow not treated.	Chronic metritis with destruction of the mucosa and calcification. Salpingitis

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2 × 1·5 × ·75 cm.	Weight	1·0 gm.
Adrenal—Right, 4 × 3 × 1·25 cm.	,,	7·0 gms.
Left, 4·5 × 2·5 × 1·25 cm.	,,	8·0 ,,
Epiphysis, 1 × ·5 × ·5 cm.	,,	0·3 gm.

Thyroid—Right half, 4 × 3·5 × 1 cm.
Left half, 3·5 × 3·5 × ·8 cm.
Length, including isthmus, 17 cm., weight 13·5 gms.
Parathyroid, 1 cm.

CASE 10. (See Appendix II, Fig. 10.)

FRIESLAND COW, 10 YEARS OLD.

Date of first examination, 12.8.25. *Date of slaughter*, 17.8.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1.260 lb. The head is coarse, Neck is short and thick. Shoulders coarse. There is a hollow in the back at lumbo-sacral junction. The pelvic ligaments are relaxed. The cow has lactated, but is now dry. No. of calves—1. Date of parturition—17.6.19. Little is known of the cow prior to her arrival at this Institute. Since her arrival, between August and December, 1925, oestral periods were frequent and irregular. The cow showed marked symptoms of nymphomania. Between January and August, 1926, there were two oestral periods, namely, in February and July. The animal continued to show symptoms of nymphomania, even though she would not allow herself to be served. Contagious abortion test—negative. Tuberculin test—negative.	Vulva normal. Vagina normal. Cervix normal is size and consistency; mucosa normal. Ostium uterinum is easily penetrated with the small cervical dilator. The uterus and uterine horns are partly abdominal, normal in size and consistency. Both fallopian tubes are normal. Left ovary size of a walnut, firm in consistency. No evidence of ripening Graafian follicle. Right ovary size of a pigeon's egg, contains two prominent corpora lutea, each about ·5 cm. in diameter. Broad ligaments are normal. *Diagnosis*: retained corpus luteum in right ovary. In November, 1925, a cyst developed in the right ovary. In February, 1926, degeneration of the uterine wall could be diagnosed per rectum and a second cyst developed in the right ovary. In April both ovaries were cystic and the uterine wall more flaccid. In August both ovaries were again cystic.	Vulva and vagina normal. Cervix, 10×4·5 cm.; width of wall, 2·2 cm.; mucosa pale pink. Ostium uterinum contains a small quantity of tenacious mucus. Uterus, 9×4·5 cm.; width of wall, ·4 to ·3 cm.; right horn, 17×2·5 cm.; width of wall, ·6 to ·4 cm. The uterus is flaccid on palpation and thinwalled. The horns are markedly tapering towards the anterior extremity. The serosa is smooth, yellowish grey in colour. The uterus contains a small quantity of tenacious slightly opaque mucus. The mucosa of the right horn is of a greyish-pink colour and shows numerous small blood-vessels. The cotyledons are reduced in number and are not prominent. There is an entire absence of cotyledons in the mucosa of the pars indivisa. The left horn shows a few small cystlike structures up to 2 mm. in diameter. These contain a clear fluid. The right horn is pale yellowish-grey in colour. The cotyledons are few in number and reduced in size. The fallopian tubes appear normal in size. The broad ligaments are normal.	Left ovary, 4× 2·8×2·5 cm. It is oval in shape. On palpation it is fluctuating. There are a few small cyst-like structures present underneath the capsule; these vary up to ·4 cm. in diameter. At the middle of the free border there is a large cyst-like prominence, 1·6 cm. in diameter, over which the capsule is transparent. On section, the ovary contains two cyst-like structures, 1·6 and 1·3 cm. in diameter. These contain a clear slightly yellowish fluid. The remainder of the ovary is greyish-white in colour and firm in consistency. Traces of atrophied corpora lutea are present. No recent corpus luteum is present. Right ovary, 5·4 3·4×3 cm. On palpation it is fluctuating. Some small cyst-like structures, about ·4 cm. in diameter, are apparent through the surface. There is a large prominent cyst 3 cm. in diameter over which the capsule is transparent. On section, the ovary contains three cyst-like structures, the largest of which is 3 cm. in diameter. These contain a clear fluid. The cysts are separated by very thin septa. The ovarian tissue is greyish-white in colour and firm in consistency. Traces of atrophied corpora lutea can be seen, but no recent corpus luteum is present.	*Adrenal glands.*— Right—hyperaemia of cortex. Left—hyperaemia of cortex. *Pancreas.*—Normal. *Thymus.*—Normal. *Epiphysis.*—Normal. *Hypophysis.*—Hyperaemia of the whole gland, especially the pars distalis. *Supramammary lymph gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Metritis chronica catarrhalis with commencing cystic formation. Marked enlargement of the vessels in the zona muscularis. Many wandering cells in the mucosa. *Fallopian tubes.*—Villi thickened by fibrous tissue. Epithelial cells unchanged. *Ovary.*— Right and Left—Graafian follicle cysts. *Mammary Gland*—Normal. *Thyroid Gland.*—Normal.	12.8.25: 2 Corpora lutea expressed from right ovary; this was followed by weekly ovarian and uterine massage. 13.11.25: Cyst expressed from right ovary. Massage continued. Served 25.11.25 and 8.2.26. 10.2.26: Cyst ruptured right ovary. 12.4.26: Cyst ruptured both ovaries, followed by uterine irrigation with Lugol's iodine, and pituitary infundibilular extract 1½ c.c., adrenalin 2 c.c. subcutaneously. 2.7.26: Served. 5.8.26: Case considered hopeless.	Retained corpus luteum followed by cystic oophoritis. Microscopic examination showed metritis chronica catarrhalis, with some atrophy of the uterine wall. There is not much doubt that this was the original lesion, although it was not diagnosed clinically.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis. 1·2 × ·3 × ·3 cm.	Weight 0·2 gm.
Hypophysis. 1·8 × 1·5 × 1·3 cm.	,, 3·5 gms.
Adrenals—Right, 6·2 × 3·8 × 1·2 cm.	,, 15·5 ,,
Left. 4·3 × 4 × 1·8 cm.	,, 15·0 ,,

Thyroid—Right half, 6 × 3 × 1 cm.
Left half, 6 × 3·5 × 1·2 cm.
Length, including isthmus, 25 cm., weight 25 gms.

CASE 11. (See Appendix II, Fig. 11.)

FRIESLAND COW 1632, AGE ABOUT 7 YEARS.

Date of first examination, 12.8.25. *Date of slaughter*, 25.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,220 lb. Head is coarse. Slight depression in the region of the lumbo-sacral junction. The pelvic ligaments are slightly relaxed and the root of the tail somewhat elevated. The udder appears to have lactated, but is now dry. There is no history as to whether this cow was ever pregnant. During the six months previous to examination there was no history of oestrum. While she was under observation at this Institute for eight months, she showed oestrum on one occasion only. Contagious abortion test, negative. Tuberculin test, negative.	The vulva does not admit the hand; the mucosa is normal. The vagina when examined from the rectum appears normal. The cervix appears somewhat enlarged and abnormally firm. The uterus is abdominal; it hangs well forward in the abdominal cavity; its wall is thin and atonic; it is fluctuating and the horns seem almost equal in size; it appears to contain from 1–1½ litres of fluid. The walls are smooth, no cotyledons can be felt. The size and pulsation of the uterine arteries are unchanged. Both fallopian tubes appear normal. The right ovary is normal in size; it contains the corpus luteum of the last interovulation period. The left ovary is normal in size and firm in consistency; no developing Graafian follicles can be felt. *Diagnosis:* hydrometra, degeneration of the uterine wall. While under observation, the fluid in the uterus accumulated somewhat, and after ovulation on the 22nd May, 1926, a corpus luteum developed in the left ovary.	The vulva admits three fingers; mucosa normal. There is evidence of a cicatrix close to the dorsal commissure. The vagina contains clear tenacious mucus; mucosa pale. Cervix. 10 × 7·5 cm.; width of wall, 3·75 cm. The pars vagina is pinkish-red in colour and normal in appearance. On section, the cervix cuts abnormally firm. The transverse folds are very well developed. Between the third and fourth fold there is a dialatation, 4 × 3 × 2 cm. It contains clear tenacious mucus. It is lined by pale mucosa, which shows slight anteroposterior ridging. The cavity is in communication with the uterus through an opening which admits a small probe only. The uterus is 16 × 10 cm.; width of wall, ·5 cm., right horn, 19 × 5 cm.; left horn, 26 × 4 cm.; width of wall of horns, ·5–·3 cm. The uterus is flaccid and thin-walled; its serous surface is smooth, greyish-pink in colour. On section, it contains 2 litres viscid fluid. Mucosa is pale pinkish-grey throughout; there is no evidence of cotyledons. The left horn shows three cysts and the right horn one cyst. These vary in size from 1·6–1 cm. in diameter. They are prominent and stand above the surrounding tissue. On section, they contain an opaque fluid. The cysts appear to be confined to the mucous surface. The fallopian tubes appear normal in size and consistency. The fimbriated extremity is also normal. The broad ligaments are elongated.	Right ovary, 3·4 × 2 × 2 cm. It shows on its surface several follicles, varying in size from ·2 mm. to 1·8 cm., over which the capsule is semi-transparent. The surface also shows four small areas of lutein tissue, the remains of atrophied corpora lutea. On palpation the ovary is firm. On section the ovarian tissue appears normal. There are several Graafian follicles in various stages of development. There are four atrophied corpora lutea present, the largest being ·5 cm. in diameter. Left ovary, 2·5 × 1·8 × 1·3 cm. Its surface also presents several small follicles, up to ·5 cm. in diameter, over which the capsule is semi-transparent. There is also a prominent corpus luteum present. The surface shows five atrophied corpora lutea of previous interovulation periods. On palpation, the ovary is firm. On section, the ovarian tissue appears normal in colour and consistence. There is a large pale yellow corpus luteum present, 1·8 × 1·5 cm. in diameter.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Pancreas.*—Normal. *Adrenals.*— Right, normal. Left, normal. *Thymus.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Some increase in the interstitial connective tissue. *Vagina.*—Normal. *Cervix.*—Dilated portion, fibromuscular wall lined with cells heavily charged with mucus. *Uterus.*—Atrophy of the muscularis, cystic enlargement of some of the mucous glands. *Fallopian tubes.*—Normal. *Ovary.*— Right and Left—many attetic follicles, also normal follicles. Corpus luteum shows increase in connective tissue stroma.	Case considered hopeless. No treatment tried. The cow was kept under observation to observe oestrum and interovulation periods.	Hydrometra. Degeneration of the uterine wall. Deformity of the cervix.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis,	1·3 × ·4 × ·4 cm...........	Weight 0·3 gm.
Hypophysis.	2·6 × 1·7 × 1 cm......	,, 3·0 gms.
Adrenals—Right,	·9 × 3·5 × 1·3 cm....	,, 15·0 gms.
Left,	5·2 × 3·8 × 1·6 cm.....	,, 13·5 ,,

Thyroid—Right half, 5·7 × 6·2 × 6 cm.
Left half, 4·5 × 3·7 × ·8 cm.
Length, including isthmus, 23 cm., weight 21·5 gms.

CASE 12. (See Appendix II, Fig 12.)

FRIESLAND COW 1633, 6 YEARS OLD.

Date of first examination, 12.8.25. *Date of slaughter*, 26.11.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,210 lb. The animal presents a normal appearance and has lactated. The udder is now dry, but is soft and pliable. There are no signs of nymphomania. No. of calves, 1. Date of last parturition, unknown. Contagious abortion test, negative. Tuberculin test, negative.	Vulva admits the hand with difficulty. The vagina is normal. The cervix does not show any change. The uterine body and horns are slightly flaccid. Immediately in front of the bifurcation of the uterus there is a hard body which lies on the abdominal floor, size 10 × 6 × 4 cm. It feels like fatty tissue. Similar firm bodies can be felt in the omental fat. Fallopian tubes are enlarged and tortuous. The left is difficult to examine throughout its entire length, on account of being involved in the tumour mentioned. Right ovary, size of hen's egg; it is fluctuating. The fimbriated extremity of its tube is attached, enlarged, and cystic. Left ovary, size of hen's egg; it is fluctuating. It contains a large cyst. Broad ligaments—Right normal; left has portion of the above-mentioned growth attached. *Diagnosis:* Hydrosalpinx, cystic oophoritis, new growth.	Vulva and vagina are normal. The cervix is normal in appearance. The uterus: attached to the broad ligament on the left side close to the attachment of the left horn and body of the uterus is a greyish-yellow hard body which on section cuts like fat. It has a mottled appearance on section, greyish-white and greyish-yellow in colour and somewhat opaque. On opening the uterus, the mucosa appears somewhat swollen. There is a small accumulation of mucous material. Fallopian tubes are enlarged, convoluted, and bosselated. They gradually increase in diameter towards the ovary, up to 1 cm. The fimbriated extremity of the tubes are attached to the ovary and are filled with a semi-transparent liquid. The tubes on section contain a clear fluid and the walls are very thin.	Left ovary, 7 × 4½ × 4½ cm. There is cystic degeneration. There are some reddish fibrous filaments attached to its surface. The fimbriated extremity of the fallopian tubes is attached all round its border. On section, there is a large cyst, 5 cm. in diameter, containing a slightly opaque yellowish fluid. It is surrounded by a thin capsule of fibrous tissue. There is a small triangular portion of greyish tissue remaining from the normal ovary and in this there is a triangular cavity which contains brownish material. Right ovary, 7 × 5 3½ cm. There is cystic degeneration. It is also covered irregularly with reddish fibrous filaments. The fimbriated extremity of the fallopian tube is attached all round its border. On section, it contains two large cysts, 4 and 3 cm. in diameter. These fill practically the whole ovary; they contain a slightly opaque yellowish fluid. Each cyst is covered only by a thin wall of fibrous tissue. There is only a very small triangular portion of the greyish tissue remaining from the normal ovary.	*Thyroid gland.*—Normal. *Epiphysis.*—Several corpora amylacea, some very large. *Hypophysis.*—Normal. *Cervix.*—Normal. *Uterus.*—Metritis chronica catarrhalis. *Fallopian tubes.*—Hydrosalpinx and perisalpingitis. *Ovary.*— Left and Right.—large cysts with connective tissue wall. *Mammary gland.*—Normal. New growth on broad ligament—lipoma.	Prognosis hopeless. No treatment tried.	Hydrosalpinx. Cystic degeneration of the ovaries. Metritis chronica catarrhalis (evident at histological examination only.

SIZE AND WEIGHTS OF ENDOCRINES.

Adrenals—Right, 6 × 3·5 × 1 cm....... Weight 15·5 gms.
Left. 4 × 4 × 1·5 cm......... ,, 12·5 ,,

Thyroid—Right half, 3·5 × 7 × 1 cm.
Left half, 3·5 × 6 × 1 cm.
Length, including isthmus, 21 cm., weight 26·5 gms.

CASE 13. (See Appendix II, Fig. 13.)

FRIESLAND COW 1636, 9 YEARS OLD.

Date of first examination, 12.8 25. *Date of slaughter*, 19.8.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 998 lb. The cow is normal in appearnace. The udder is small, but appears to have lactated. The mammary tissue is soft, pliable. No. of calves, 1. Date of parturition, 19/9/19. Prior to arrival at this Institute, there is no record of the nature of oestrum; the cow had been bred several times without result. Between August, 1925, and January, 1926, the oestral periods were normal. There was complete absence of oestrum between January and June, 1926. In June and July there were two periods of oestrum. There was no evidence of nymphomania. Contagious abortion test, negative. Tuberculin test, negative.	Vulva is small and admits only two fingers. Mucosa is pale and shows a few elevated nodules. Vagina from the rectum appears normal. Cervix appears slightly small, but is normal in consistency Uterus and uterine horns are small, short, and entirely pelvic; consistency normal. Both fallopian tubes are unchanged. Right ovary is the size of a hen's egg; normal in consistency; contains a prominent corpus luteum. Left ovary size of a walnut; normal in consistency; no evidence of a developing Graafian follicle on palpation. Broad ligaments normal. *Diagnosis:* corpus luteum persistens. While under observation the right ovary became cystic in February, 1926. In April both ovaries became cystic and the udder began to show signs of lactation. The cow began to show evidence of nymphomania with relaxation of the pelvic ligaments. In August, 1926, the ovaries were again cystic, and it was decided to destroy the animal.	The vulva and vagina are normal. Cervix, 7 × 3 cm.; width of wall, 1·5 cm. Mucosa pale pink in colour The ostium uterinum contains a small quantity of clear mucus. Uterus body, 6·5 × 3 cm.; width of wall, ·6 cm.; both horns, 17 × 2 cm.; width of wall, ·6 × ·8 cm. Uterus and uterine horns are small in size and flaccid. The horns are markedly tapering towards their anterior extremity. The serosa is yellowish-pink in colour and smooth. The mucosa is greyish-pink in colour, smooth, moist, and perhaps slightly swollen. There is little evidence of cotyledons and those that remain are of a yellowish colour. The muscular coat is exceedingly thin, about 2 mm. in diameter. There is a small quantity of clear tenacious mucus present. Both fallopian tubes are normal. Post-mortem showed tubercular lesions in the bronchial lymph glands.	Right ovary, 5·5 × 3·5 × 3·4 cm. There are traces of two corpora lutea on the surface. On palpation it is cystic throughout and in parts the capsule is exceedingly thin and transparent. On section, the ovary contains four cyst-like structures, the largest of which is 2·6 cm. in diameter. The capsule is smooth. The contents are transparent and slightly yellowish; thin liquid in consistency. There is little ovarian tissue remaining. It is greyish-white in colour and firm in consistency. There are traces of atrophied corpora lutea. The ovary has not recently ovulated. Left ovary, 4 × 2·6 × 2·3 cm. The surface is mostly transparent. In parts one can detect the presence of cyst-like structures through surface. These vary in size. On palpation the ovary is fluctuating and its capsule for the most part is thin and transparent. On section, the ovary is almost entirely filled with three cyst-like structures. The largest is 2·4 cm. in diameter. There is little ovarian tissue remaining. It is greyish-white in colour, and firm in consistency. The liquid contents of the cysts are transparent and slightly yellowish. There are traces of atrophied corpora lutea present. The ovary has not recently ovulated.	*Adrenals.* Left and Right.—Hyperaemia of the cortex. *Epiphysis.* Normal. *Hypophysis.* Normal. *Thymus.* Increase of the insterstitial connective tissue. Infrequency of Hassal's corpuscles. *Pancreas.* Normal. *Thyroid.* Normal. *Supramammary lymph gland.* Normal. *Mammary gland.* Evidence of lactation. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Slight oedema. Local enlargement of the mucous glands. Atrophy of the muscularis. *Fallopian tubes.*—Normal. *Ovaries.* Left and Right. Cystic degeneration of the Graafian follicle, oedema, hyperaemia, atretic follicles.	12/8/25: Corpus luteum expressed from right ovary. 11/2/26: Cyst expressed from right ovary. 7/4/26: Cysts pressed from both ovaries. 7/5/26 to 7/6/26: Subcutaneous injection of pituitary infundibular extract 2 c.c., adrenalin 1 c.c., twice weekly. Bred several times without result.	Corpus luteum persistens, followed by cystic ovaritis.

SIZE AND WEIGHTS OF ENDOCRINES.

Eypiphysis, 1·5 × ·4 × ·4 cm..........	Weight 0·15 gm.
Hpophysis, 2 × 1·8 × 1·2 cm..........	,, 2·5 gms.
Adrenals—Right, 4·2 × 4·6 × 1·9 cm...	,, 14·0 ,,
Left, 5·5 × 3·2 × 1·5 cm.....	,, 17·0 ,,

Thyroid—Left half, 4·8 × 5·5 × ·6 cm.
Right half, 5·6 × 3 × 1 cm.
Length, including isthmus, 27 cm., weight 20·5 gms.

CASE 14. (See Appendix II, Fig. 14.)

FRIESLAND COW 1637, 5 YEARS OLD.

Date of first examination, 12.8.25. *Date of slaughter*, 29.10.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,520 lb. The general appearance is somewhat ox-like. The head is coarse. The pelvis is somewhat small in proportion to the cow's enormous frame. The udder has not lactated. The animal has never shown oestrum. Contagious abortion test, negative. Tuberculin test, negative.	The vulva shows a few dormant nodules of granular vaginitis. It does not admit the hand. On introducing two fingers to a depth of about 4 inches the vulva is found to end in a *cul-de-sac*. Per rectum the vagina and cervix appear to be undeveloped. The uterus cannot be easily palpated. It is resprsented by a firm, small flattened cord-like structure which is not compressible. The uterine horns are small and firm. Attached to the posterior extremity of the cord-like uterus there are two small elongated projections, about 2½ inches in length, extending in an outward and forward direction. These are also very firm and cord-like. Fallopian tubes cannot be felt. Both ovaries are small and undeveloped. They are difficult to examine owing to the very high condition of the animal. Broad ligaments are short.	The vulvar mucosa shows a few small yellowish semi-transparent nodules. It ends in a blind extremity, and is only 10 cm. long. The vagina is absent. The cervix is also undeveloped. The uterus is small and undeveloped. Left cornu, 7 × 1·5 cm.; right cornu, 8 × 1·5 cm.; body of uterus, 16·5 × 2 cm. The whole structure is closely surrounded by loose connective tissue. On section, there is a small lumen lined with mucous membrane. The lumen is somewhat sacculated and extends from the apices of the horns to the projections at the posterior extremity of the structure. It varies from 2 to 4 mm. in diameter. It has no communication with the two appendices at the base of the uterus, nor is there any communication with the vulva. The two appendices are 8 cm. long and 1 cm. in thickness. They end anteriorly in a rounded extremity. Posteriorly they touch the uterus on either side. On section, they are lobulated and gland-like. Fallopian tubes not developed.	Right and left ovary are similar in appearance, 1·5 × 5 cm. × 2 mm., light brownish in colour. At the pole nearest the uterine horn there is a cyst 4 mm. in diameter. In the left ovary this cyst-like structure is placed somewhat nearer the centre.	*Hypophysis.*—Normal. *Epiphysis.*—Normal. *Thyroid.*—Vesicles irregular. Some large and filled with colloid, others small. *Adrenal.*—(1) Normal; (2) Normal. *Supramammary lymph gland.*—Normal. *Mammary Gland.*—Isloated islets of mammary parenchyma in a thick fibrous and fatty stroma. Some of the vesicles filled with a fluid staining red with eosin. *Wolffian ducts.*—Muscular and connective tissue wall lined with cubical epithelium. *Uterus.*—Shows mucous glands. Cylindrical epithelium. Glands less numerous than normal. *Ovaries.*—Both show a small cyst-like structure with a fibrous tissue wall. No follicles present.	Prognosis hopeless. No treatment.	Arrest in the development of the genital tract. "Neuter."

SIZE AND WEIGHTS OF ENDOCRINES.

Adrenals—Right, 5·75 × 4 × 1·25 cm... Weight 13·0 gms.
Left, 5 × 3·5 × 1·25 cm..... „ 12·0 „

Thyroid—Right half, 4 × 5 cm.
Left half, 4 × 4·5 cm.
Length, including isthmus, 19·5 cm., weight 22·5 gms.

CASE 15. (See Appendix II, Fig. 15.)

FRIESLAND COW 1638, 7 YEARS OLD.

Date of first examination, 12.8.25. *Date of slaughter*, 22.4.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,850 lb. The appearance of the head is somewhat coarse, otherwise the animal is normal. The udder is dry, but the animal has lactated. No. of calves, 2. Date of last parturition, 21/1/23. This animal was reported to have shown regular oestral periods, but while under observation the oestral periods were very irregular. The cow has never aborted and there was no evidence of contagious granular vaginitis. Contagious abortion test, positive. Tuberculin test, negative.	The vulva is normal. The vagina contains a small amount of thick tenacious mucus. The cervix shows a somewhat ragged posterior extremity, but the mucosa is normal in colour. The os uterus admits the point of the little finger to the depth of 1 inch. Per rectum the cervix shows a decreasing diameter towards its anterior extremity. It seems normal in consistency. The uterine body and horns are mostly abdominal. The horns hang well forward in the abdominal cavity; they are flaccid and somewhat enlarged. The left fallopian tube appears normal. The right fallopian tube is difficult to palpate at its distal extremity as it is involved in a connective tissue formation. The uterus shows some loose adhesions with its neighbouring pelvic organs. The right ovary is normal in size, but its fimbriated extremity is adherent and contains a soft cyst. The left ovary is normal in size and consistency. The broad ligaments appear normal. *Diagnosis:* salpingitis of the right fallopian tube and pavilionitis, metritis chronica catarrhalis.	The vulva and vagina present some small red spots; the mucosa is otherwise pale. The cervix shows some small red spots and slightly protruding first fold; length 8 cm., width 5 cm. The mucosa is pale; the second fold shows a dark red area. The os uterus is exceedingly small at its cranial opening, being only a couple of millimetres in diameter. The uterus body is 13 cm. long; right horn, 18 cm.; left, 19 cm.; width of body 8 cm.; of horns, 4 cm. The serous surface of the body shows numerous fibrous filaments, through which it is loosely adherent to the neighbouring structures of the pelvis; these adhesions are confined to the dorsal surface and to the ligamentum intercornualia. The wall of the uterus is 1·4 cm. thick; it is greyish-yellow in colour and normal in consistency: the mucosa is reddish-grey in colour, it is soft and very moist. There is no trace of normal cotyledons. Along the ridges several small depressions can be seen, circular in outline, from 4 to 6 mm. across. The left fallopian tube is unchanged. Its fimbriated extremity is somewhat thickened. The right fallopian tube appears somewhat enlarged at its cornual extremity. The fimbriated extremity of the fallopian tube encapsules the ovary, to which it is firmly adherent; it is thickened and fibrous; its cavity contains a quantity of clear fluid. The fallopian tube behind the pavilion is tortuous and difficult to follow as it is embedded in fibrous tissue.	Left ovary, 4 × 3 × 2·5 cm. It presents numerous Graafian follicles in different stages of development; it contains the corpus luteum of the last intervoluation period. On section there is a corpus luteum 2·3 × 2cm. in diameter, and a Graafian follicle ·5 cm. in diameter. There are five atrophied corpora lutea present. The remainder of the ovary is reddish-grey and normal in consistency. Right Ovary, 3·2 × 1·7 × 1·6 cm., firmly attached to fimbriated extremity of the fallopian tube, which is closed around it. On section, the ovary is fibrous, greyish-white in colour, with several bloodvessels. There are three atrophied corpora lutea present. There is a cyst-like structure, 1·8 cm. in diameter, towards its centre, and a small cavity, ·5 cm. in diameter, filled with what appears to be clotted blood.	*Adrenal.*— Right and Left.—Large granules of brownish-yellow pigment in the zona glomerulosa. *Epiphysis.*—Corpora amylacea very frequent. *Hypophysis.*—Hyperaemia in pars distalis. *Thyroid.*—Normal. *Pancreas.*—Normal. *Thymus.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Numerous concretions and extensive fibrosis. *Vagina.*—Normal. *Cervix.*—Fibrosis and slight polypus formation. *Uterus.*—Metritis chronica catarrhalis, perimetritis. *Fallopian tube.*— Right.—Salpingitis, pavilionitis, perisalpingitis. *Ovary.*— Left.—Normal corpus of luteum. Normal follicles and atretic follicles. Right.—Cyst with fibrous wall, fibrous filaments on surface.	No treatment was carried out. The case was considered hopeless, and the animal was kept under observation to watch the oestrous cycle.	Pavilionitis, salpingitis, (right side), metritis chronica catarrhalis, cystic oophoritis.

Size and Weights of Endocrines.

Epiphysis, 1·7 × ·7 × ·7 cm........... Weight 0·35 gm.
Hypophysis, 2·1 × 1·6 × 1·5 cm........ „ 2·8 gms.
Adrenals—Right, 6·3 × 4·2 × 1 cm..... „ 19·5 „
Left, 5 × 4·8 × 1·3 cm,...... „ 18·0 „

Thyroid—Right half, 4 × 6 × ·8 cm.
Left half, 3·7 × 6 × ·8 cm.
Length, including isthmus, 23 cm., weight 24·5 gms.

CASE 16. (See Appendix II, Fig. 16.)

FRIESLAND COW 1639, 9 YEARS OLD.

Date of first examination, 12.8.25. *Date of slaughter*, 12.3.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,550 lb. The head is coarse and ox-like. The external angle of the ilium on the left side is badly depressed, as the result of an old fracture, causing marked deformity of the pelvis. There is slight left lateral curvature of the spine. There is a marked sinking of the spine in the region of the lumbo-sacral junction. Relaxation of pelvic ligaments is pronounced. The root of the tail is elevated. There are pronounced symptoms of nymphomania. No. of calves, 4. Last parturition, 27/9/22, since when periods of oestrum have been irregular. Contagious abortion test, negative. Tuberculin test, negative.	The vulva and vagina are normal. The cervix shows a normal posterior opening, but it seems abnormally firm in consistency. The uterus is partly abdominal; it is normal in size and consistency. Both horns of the uterus are soft, especially towards their apices. Both fallopian tubes are normal in size. Right ovary is the size of a small hen egg. It is fluctuating at its anterior and posterior extremities. Left ovary similar in size. It is lobulated and fluctuating. The broad ligaments are normal. *Diagnosis.*—Cystic oophoritis, metritis chronica catarrhalis. The cow was examined on 28/9/25, 6/11/25, 21/11/25, 15/1/26, 11/2/26. The cysts again developed in the ovary after rupture, and the uterine wall became very flaccid and atonic.	Vulva shows nothing unusual. The vagina contains a small quantity of opaque mucus. The cervix shows a somewhat ectropic posterior fold. On section, the mucosa is slightly reddish, and the second and third folds are not well marked. The uterine body is 9 cm. long; horns 20 cm. long; it is flaccid. Towards the apex of each horn a few cyst-like prominences are seen. On section, the mucosa, especially towards the apices of the horn, shows numerous cyst-like elevations, which vary in size from a pinhead to a small marble. These, on section, contain a clear, slightly viscid fluid. Cysts are also found in the uterine wall: the larger ones pass through the wall and appear on the serous surface. The small ones are confined to the mucosa. The cotyledons on section are reddish in colour. The mucosa of the uterus is of a yellowish-grey colour except over the cotyledons, where it is reddish-grey. Both fallopian tubes appear normal. Broad ligaments appear normal.	The left ovary 4 × 3·7 × 2 cm. It presents two large cystic prominences, which are slightly compressible and fluctuating; the remainder of the ovary is firm. On section, towards its surface there are three cyst-like structures varying in size from 1 to 2 cm. in diameter. The two smaller cysts contain clear yellow fluid, the large cyst contains blood-stained fluid, and at one part there is a local lining of lutein tissue. In addition, there is a small cavity 1·5 cm. in diameter which contains clotted blood; this is apparently the remains of a cyst which was ruptured 11/2/26. The remaining part of the ovary is whitish-grey in colour. Right ovary, 4 × 2·5 × 2 cm. It presents two cystic prominences which are slightly compressible and fluctuating. On section, it contains three cyst-like structures, varying in size from 0·7–1·7 cm.; these are placed towards its surface. On section, they contain a clear yellow fluid. There are also present some smaller cyst-like structures. The remaining part of the ovary is greyish-white in colour and firm. There is no recent corpus luteum in either ovary.	*Adrenals.*— Right and Left.—Normal. *Pancreas.*—Normal. *Thyroid.*—Normal. *Hypophysis.*—Normal. *Epiphysis.*—Corpora amylacea. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Metritis chronica catarrhalis cystica. *Fallopian tubes.*—Normal. *Ovary.*—Normal and atretic follicles. Small cystic degeneration. Fibrous filaments on the surface. Traces of corpus albicans with some lutein cells interspersed in the fibrous tissue.	12/5/28: Cysts expressed and uterine wall massaged. Uterine irrigation and massage was continued up to the time when prognosis was considered hopeless, 11/2/26.	Metritis chronica catarrhalis cystica

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis..................................	Weight 0·2 gm.
Hypophysis, 2 × 1·6 × 1·6 cm..........	„ 3·8 gms.
Adrenals—Right, 8 × 5 1·6 cm.......	„ 40·9 „
Left, 5·9 × 5·6 × 1·7 cm.....	„ 38·2 „

Thyroid—Right half, 9 × 1·4 cm.
Left half, 9 × 1·6 cm.
Length, including isthmus, 25 cm., weight 87·5 gms.

CASE 17. (See Appendix II, Fig. 17.)

FRIESLAND COW 1640, 14 YEARS OLD.

Date of first examination, 12.8.25. *Date of slaughter*, 29.10.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,350 lb. There is a fracture of the external angle of the ilium on the left side. There is a marked depression at the lumbo-sacral junction and a relaxation of the pelvic ligaments with elevation of the root of the tail. The udder is normal and non-lactating. No. of calves, 6. Date of last parturition, 10/7/20. Oestral periods have been irregular and at very frequent intervals. The cow has been a typical nymphomaniac for a considerable period. Contagious abortion test, negative. Tuberculin test—negative.	The vulva shows a normal mucosa. The vagina contains a quantity of opaque tenacious material with some yellow flocculi. The posterior opening of the cervical canal has somewhat ragged lips. The cervical canal admits the first finger easily for the distance of 2 inches. The mucosa of the first fold is slightly reddish in colour. The cervix is slightly flattened from above to below, and appears to be very firm on palpation. The uterus is mostly abdominal; it is somewhat enlarged and flabby with thin walls. A cyst-like prominence can be felt on its upper surface, somewhat posterior to the bifurcation. The right fallopian tube shows numerous cysts along its length. The left fallopian tube appears normal, except its pavilion, which is cystic. Right ovary is oval in shape, enlarged to the size of a hen's egg and fluctuating. The left ovary is somewhat larger than a hen's egg, oval and fluctuating. The broad ligaments appear unchanged. *Diagnosis*: cystic oophoritis, salpingitis, cystic degeneration of the uterine mucosa.	The cervix is 9 cm. in length. Mucosa is greyish-pink in colour and shows numerous scattered cysts up to ·7 cm. in diameter. The cysts are confined towards the mucous surface. The uterine body is 19 cm., width 9·5 cm.; and the horns 21 cm., width 7·5 cm. It is flaccid and thin-walled. Its surface presents a number of cyst-like prominences varying size from 2 mm. to 3·5 cm. in diameter. Over the horns the cysts lie close together; they are filled with a transparent fluid. The serosa of the remaining portion is of a yellowish-pink colour. In the hrons the lymph vessels are swollen. The mucosa presents a greyish-yellow colour; it is studded throughout with numerous cysts varying in size from a pin's head to a marble; one large cyst is 3·5 cm. in diameter. These cysts are confined mostly towards the mucous surface, but the larger cysts pass completely through the walls. The cotyledons are not prominent, and are greyish-yellow in colour. Right fallopian tube shows cysts at intervals along its length, varying in size up to a pea. Its pavilion is attached to the ovary and is cystic. Left fallopian tube appears normal, but its pavilion is adherent and cystic. The broad ligaments appear normal.	Right, 6 × 4·5 × 3 cm. It is oval and cystic in character. The fimbriated extremity of the fallopian tube is attached. Ovary fluctuating. On section, there are five well-developed central cysts varying in size from ·75 to 2·5 cm. in diameter. These contain a clear slightly yellowish fluid. The normal ovarian tissue has atrophied, only a small portion of greyish tissue remaining. There is no evidence of corpora lutea. Left, 5·5 × 4·2 × 2·5 cm. It is oval and fluctuating. The fimbriated extremity of the fallopian tube is attached and slightly cystic. On section, there are eight cysts present, varying in size from ·5 to 2·5 cm. in diameter. These cysts cause atrophy of the normal ovarian tissue, only a small shell of greyish tissue remaining.	*Epiphysis*.—Normal. *Hypophysis*.—Normal. *Adrenals*.—Both show some increase in the connective tissue stroma, especially marked in the medulla and zona reticularis. *Thyroid gland*.—Normal. *Pancreas*.—Normal. *Supramammary lymph gland*.—Fibrosis. *Mammary gland*.—Concretions. *Cervix*.—Cystic formation in the mucosa. *Uterus*.—Metritris chronica catarrhalis cystica. *Fallopian tubes*.—Hydrosalpinx and pavilionitis. *Ovaries*.—Both show cystic degeneration, cyst wall of fibrous tissue without lutein cells. *Parathyroid*.—Shows numerous vesicles lined with a cubical epithelium. Many of the vesicles are filled with colloid. These vesicular areas are surrounded by a thick connective tissue layer.	Prognosis hopeless. No treatment tried.	Cystic oophoritis, pavilionitis, salpingitis, cystic degeneration of the cervical mucosa, metritis chronica catarrhalis cystica.

SIZE AND WEIGHTS OF ENDOCRINES.

Adrenals—Right, 7 × 5·4 × 2·5 cm..... Weight 35·5 gms.
Left, 7 × 5·8 × 2·2 cm,....., „ 27·75 „

Thyroid—Right half, 5 × 7 cm.
Left half, 4·5 × 6 cm.
Length, including isthmus, 23 cm., weight 39·5 gms,

CASE 18. (See Appendix II, Fig. 18.)

FRIESLAND COW 1657, 6 YEARS OLD.

Date of first examination, 22.8.25. *Date of slaughter*, 20.4.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus. Genital Tract.	Post-mortem Examination of the Genital Apparatus. Ovaries.	Microscopic Examination.	Treatment.	Cause of Sterility.
Condition good. Weight 1,210 lb. The udder is in the later stages of lactation. The cow has had a muco-purulent discharge from the vulva for some months, which became very profuse a few weeks previous to examination. No. of calves, 1. Date of parturition, April, 1923. Since parturition periods of oestrum have been irregular. The cow has suffered from contagious granular vaginitis. Contagious abortion test, negative. Tuberculin test, negative.	Vulva shows dormant lesions of contagious granular vaginitis. The vagina contains a small amount of thick yellowish muco-purulent material. The posterior extremity of the cervix is dark reddish in colour and the first fold is protruding. The ostium uterinum externum admits one finger for a distance of about 4 cm. The body and horns of the uterus are somewhat enlarged and flaccid. The fallopian tubes appear normal. The right ovary is normal in size and contains a prominent corpus luteum. The left ovary is the size of a broad bean, firm; no developing Graafian follicle can be felt. The broad ligaments are normal. During treatment the body of the uterus at the point of bifurcation became abnormally hard; this condition gradually extended over the uterine horns. A large hard tumour, the size of a hen's egg, developed in the ventral wall of the right horn. The fallopian tubes became somewhat enlarged and difficult to palpate throughout. A hard body slowly developed on either side of the uterus in the broad ligaments. *Diagnosis*: Endometritis, retained corpus luteum, followed by sclerosis and the development of a new-growth (fibroma) in the uterine wall and the broad ligaments.	The vulva shows some nodules of granular vaginitis. The vagina contains some tenacious material, with yellow flocculi. The posterior extremity of the cervix is light-red in colour. The first cervical fold is protruding and somewhat ragged. Length of cervix, 9 cm. × 3·5 cm. The mucous lining of the os is pale pinkish-yellow in colour; it is markedly ridged. Scattered over the mucosa are some irregular, light yellowish patches, the largest of which is about 3 cm. long. The surface of these patches is roughened. On section, they extend deeply into the cervical wall; they are firm, almost cartilage hard, and are pale whitish-yellow in colour. Uterus—body, 13 × 6 cm.; left horn, 23 × 4 cm.; right, 24 × 4·5 cm. The serosa of the uterus is yellowish-red in colour; the horns are very much curled up, and the right horn shows an irregular hour-glass formation in the centre. The uterus throughout is abnormally hard with the exception of the apices of the horns and the pars indivisa, which are soft and flaccid. On section, the mucosa presents a greyish-red appearance; it is soft and very oedematous; no trace of normal cotyledons can be seen. The wall of the hard portion of the uterus varies in thickness; it cuts firm and is of a mottled yellow colour. The portion resembling the hour-glass already described in the right horn cuts exceedingly firm and is yellowish in colour. Scattered throughout the wall of the uterus there are some dark haemorrhagic areas. On the inferior wall of the pars indivisa there is an elongated irregular patch similar to those which have been described on the cervix. The left horn shows an area 3 cm. long from which the mucosa is removed, leaving a depressed yellowish-red granulating base. The fallopian tubes are thickened to 4 mm. in diameter. The fimbriated extremity of both tubes is attached to the ovary by a fibrous attachment. There appears to be no débris or liquid in the fallopian tubes or the fimbriated extremity. The broad ligaments are normal in length. Along the line of attachment to the uterus on either side are irregular elongated tumours about 12 × 4 × 3 cm. On section, tumour is firm pale whitish-yellow in colour, and it cuts almost cartilage hard. The anterior portion of the tumour on the right side is attached to the lateral and inferior walls of the uterus, of which it forms a part. This portion of the swelling is rounded, 6 cm. in diameter. On section, it presents a whitish to very pale yellow colour with some haemorrhagic areas.	The left ovary, 3 × 3 × 2 cm., oval in shape. Its surface shows the presence of some fibrous filaments; there is a developing Graafian follicle present. It contains a well-marked atrophied corpus luteum of the second last interovulation period. On section, shows a developing Graafian follicle 1·3 cm. in diameter, and three atrophied corpora lutea. The remaining portion of the ovary is whitish-grey in colour. The right ovary, 3·5 × 3 × 2 cm., oval in shape. It shows the corpus luteum of the last interovulation period. Its surface shows some fibrous filaments. On section, contains a corpus luteum 1·9 cm. in diameter; it is of an orange-yellow colour; its centre is cystic and contains a clear somewhat viscid fluid. Close to this corpus luteum is a smaller atrophied one. The remaining portion of the ovary is greyish-white.	*Adrenal glands.*—Normal. *Thyroid gland.*—Normal. *Hypophysis.*—Normal. *Epiphysis.*—Numerous corpora amylacea. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Slight catarrhal mammitis. *Vagina.*—Normal. *Cervix.*—fibrosis and ulceration. *Uterus.*—Metritis chronica catarrhalis with commencing cystic formation and myofibroma. *Fallopian tubes.*—Salpingitis chronica purulenta. *Ovary.*— Right.—Cystic corpus luteum. Some atretic follicles with proliferation of the theca.	22/8/25: Corpus luteum expressed. The animal was treated several times by uterine irrigation with 1 per cent. Lugol's iodine and swabbing of the cervix with Lugol's iodine. 10/2/26: Examination on this date showed tumour development in the uterus and ligaments. The case was considered hopeless, but was kept under observation until date of destruction.	Cervicitis, endometritis followed by salpingitis and tumour development in the uterine wall and broad ligaments.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·3 × ·4 × ·5 cm. .... Weight 0·2 gm.
Hypophysis, 2·2 × 1·1 × 1·7 cm. .... ,, 2·4 gms.
Adrenals—Right, 5·4 × 4·3 × 1·2 cm. ... ,, 13·0 ,,
Left, 4·4 × 5·5 × 1·4 cm. .... ,, 12·0 ,,

Thyroid—Right half, 4·6 × 4·8 × 1·2 cm.
Left half, 6·6 × 6 × 1 cm.
Length, including isthmus, 19·5 cm., weight 23·5 gms.

CASE 19. (See Appendix II, Fig. 19.)

FRIESLAND COW 1658, 6 YEARS OLD.

Date of first examination, 22.8.25. *Date of slaughter*, 10.8.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,311 lb. Normal in appearance. Udder is non-lactating. Mammary tissue normal. The tail and hindquarters are soiled with a purulent discharge from the vulva. No. of calves, 1. Date of parturition, May, 1922. While under observation for a period of 12 months at this Institution, the cow showed oestrum nine times at very irregular intervals. In June symptoms of nymphomania developed, rapidly followed by relaxation of the pelvic ligaments and external genital organs. The symptoms continued to become exaggerated until slaughtered. Contagious abortion test, negative. Tuberculin test, negative.	Vulvar-mucosa pale; shows some yellowish semi-transparent nodules. There is a foetid yellowish discharge. The vaginal mucosa is normal. It contains a quantity of muco-purulent material. The cervix shows a protruding first fold, which is red in colour; it is ragged, swollen, and firm. The ostium uterinum externum admits the finger for about 4 cm.; it contains some muco-purulent material. Per rectum cervix appears enlarged and firm. Uterus and horns are abdominal; both horns are enlarged, equal in size. They are flaccid, fluctuating, and appear to contain about ½ litre of thick fluid. Both fallopian tubes are normal in size. Left ovary size of a pigeon's egg, firm in consistency. Right ovary size of a small hen egg; it contains a fairly prominent corpus luteum towards its anterior pole. The posterior pole of the ovary is fluctuating and cystic. Broad ligaments normal. *Diagnosis:* cystic right ovary, pyometra, cervicitis.	The vulva shows some yellowish semi-transparent nodules; mucosa pale. Vagina contains a small quantity of clear viscid mucus. Cervix, 11 × 5 cm.; width of wall, 2·5 cm.; mucosa pale pink. First cervical fold is ectropic, swollen, and firm in consistency; second cervical fold partly protrudes through the ostium uterinum externum: it is also swollen and firm. Fourth cervical fold is not very prominent. Uterine body, 9 × 5 cm.; width of wall 1–·75 cm.; horns—right, 17 × 4 cm.; width of wall, 1–·5 cm.; left, 15 × 2·5 cm.; width of wall, ·65—·5 cm. The serosa of the uterus is smooth, yellowish-pink in colour. On section, the mucosa is greyish-yellow in colour and moist. The uterus contains a very small quantity of clear mucus. In the right horn the cotyledons are fairly prominent, measuring from 1·5–·5 cm. In the left horn they are not so prominent, varying from 1–·5 cm. In the wall of the right horn the vessels of the vascular zone appear very prominent. Both fallopian tubes and their pavilions appear unchanged. Both broad ligaments are normal. Carcase showed tubercular lesions in the bronchial and mediastinal lymph glands.	Right ovary, 5 × 4 × 3·5 cm., oval in shape. The surface is smooth and glistening. Through the capsule can be seen numerous small cyst-like structures up to ·4 cm. in diameter. These on section contain a clear slightly yellowish fluid. There are three large cyst-like prominence over which the capsule is transparent. The largest of these is 2·8 cm. in diameter. A trace of two atrophied corpora lutea can also be seen on the surface. On palpation the ovary feels cystic throughout. Section shows three cyst-like structures 1·4, 1·6, and 2·8 cm. in diameter. The contents are transparent and slightly yellowish in colour. The small cyst-like structures seen through the surface are apparently normal follicles. Several traces of atrophied corpora lutea are evident. The ovarian tissue is pinkish-grey and firm in consistency. Left ovary, 3·8 × 3 × 2·5 cm., oval in shape, surface smooth. It shows numerous small cyst-like areas through the surface, the largest of which is ·4 cm. There is a large cyst-like prominence ·7 cm. in diameter, over which the capsule is transparent. The corpus luteum of the last interovulation period is present: it is almost level with the surface of the ovary. Section shows an irregular orange-coloured corpus luteum 1 × 1·3 cm. in size. The cyst-like structure seen through the surface is apparently a normal follicle, as are the small cystlike structures on the surface. The ovarian tissue is pinkish-grey and firm in consistency.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Pancreas.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vulva.*—Normal. *Vagina.*—Normal. *Cervix.*—Cervicitis, chronica catarrhalis with fibrosis. *Uterus.*—Metritis chronica catarrhalis cystica. *Fallopian tubes.*—Normal. *Ovary.*— Right.—Cystic degeneration of the Graafian follicle. Several normal follicles. Left.—Slight fibrosis several normal follicles.	The uterus was irrigated with 1 per cent. Lugol's iodine and the os swabbed with pure Lugol's iodine, at weekly intervals, for five weeks. On two occasions, corpora lutea were expressed to bring about oestrum. In April, 1926, the right ovary became cystic; this was treated by rupture; cervix and uterus were again treated. In August, 1926, the cystic condition of the right ovary had again reappeared, and it was decided to slaughter the animal.	Cervicitis, pyometra and cystic oophoritis. During treatment cervicitis and pyometra were apparently cured, but recurrent cystic ovaritis of the right ovary persisted.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·3 × ·4 × ·4 cm.	Weight 0·3 gm.
Hypophysis, 2·1 × 1·8 × 1 cm.	,, 2·7 gms.
Adrenals—Right, 6·8 × 4 × 1·7 cm.	,, 20·5 ,,
Left, 6 × 3·3 × 1·5 cm.	,, 20·0 ,,

Thyroid—Right half, 5 × 5 × 1·4 cm.
Left half, 7 × 5 × 1·2 cm.
Length, including isthmus, 24 cm., weight 30·5 gms.

CASE 20. (See Appendix II, Fig. 20.)

FRIESLAND COW 1659, 11 YEARS OLD.

Date of first examination, 22.8.25. *Date of slaughter*, 28.4.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,200 lb. Normal in appearance. Udder lactating. Mammary tissue appears somewhat firmer than normal. The left supramammary lymphatic gland is somewhat enlarged. No. of calves unknown. Date of last parturition unknown, but must have been within previous twelve months. Periods of oestrum irregular and infrequent. Has suffered from contagious granular vaginitis. Contagious abortion test—negative. Tuberculin test—positive.	Vulva shows some old lesions of granular vaginitis. The vagina is normal. The posterior extremity of the cervix is enlarged and ragged. The cervical fold is ectropic. The os uterus admits the forefinger for the depth of 4 cm. Per rectum, the cervix appears slightly enlarged, but normal in consistency. The body of the uterus is flaccid and mostly abdominal. The right horn is larger than the left, both are flaccid. Both fallopian tubes are normal. Right ovary the size of a pigeon's egg, firm in consistency. It contains a large corpus luteum. There are some adhesions between the fimbriated extremity of the fallopian tube and the ovary. The left ovary is small, bean-shaped, and firm. There are also some adhesions with the fimbriated, extremity of the tube. Broad ligaments normal. *Diagnosis*: paviliouitis, retained corpus luteum, chronic endometritis. While under observation, this animal developed cystic ovary and salpingitis on the left side. On the right side a soft tumour developed in the broad ligament, which later became attached to the ovary and uterus by thick fibrous strands.	Vulva shows a few dormant nodules of contagious granular vaginitis. Vagina normal, mucosa pale. First cervical fold ectropic, pale in colour. Length of cervix, 6·5 cm.; width of wall, 2·6 cm.; mucosa pale, consistency normal. The uterus is red in colour with a slightly yellowish tinge-body, 12×6 cm.; width of wall 2 cm.; left hron, 18 cm.×3 cm.; width of wall, ·7 cm.; right horn, 20×4 cm.; width of wall, 1·5 cm. The serosa shows numerous fibrous filaments, especially on its right lateral aspects. Attached to the broad ligament on the right side, 6 cm. posterior to the ovary, is a swelling 4·2×3×2·5 cm. It is spongy to the feel, covered with a smooth capsule; it sends strands of loose fibrous tissue to the uterus and to the ovary. On section, it cuts doughy, dark red-brown in colour, with yellowish tinge in parts; it has the appearance of an old haemorrhage. On section, the uterus presents a mottled mucosa. The left horn is greyish-red in colour, with smooth mucosa. Cotyledons not prominent, with a pale yellowish-grey colour. The mucosa of the right horn is reddish-grey in colour, but darker than the left. The cotyledons are prominent up to 1·5 cm. in diameter; they are pale, horn is greyish-red in colour, with smooth mucosa. Cotyledons not prominent, with a pale yellowish-grey colour. The mucosa of the right horn is reddish-grey in colour, but darker than the left. The cotyledons are prominent up to 1·5 cm. in diameter; they are pale, whitish-yellow in colour, and give the mucosa a very mottled appearance. The left fallopian tube towards its fimbriated extremity is dilated and convoluted: it is ·5 to 1 cm. in diameter. The cornual extremity appears unchanged. Its fimbriated extremity is adherent to the ovary. The right fallopian tube does not appear increased in diameter; it is difficult to trace, as it is covered with fibrous thickenings; its extremity is attached to the ovary by fibrous tissue.	Left ovary, 5×4·8×3·5 cm., oval in shape. Its surface shows some fibrous filaments, but is for the most part smooth; it is markedly fluctuating. On section, contains a large cyst, which practically fills the entire ovary; it contains a clear yellow slightly viscid fluid. The cyst wall is smooth, reddish-grey in colour, The surrounding capsule is partly composed of a yellowish band, apparently corpus luteum tissue; the remaining portion of the capsule is greyish-white in colour. The normal ovarian tissue has almost undergone complete atrophy as a result of pressure from the developing cyst. Right ovary, 4×2·5×2·5 cm., shows numerous fibrous filaments, but is for the most part smooth. Some strands of fibrous tissue pass between it and the tumour described in the broad ligament. It presents a prominent corpus luteum, 1 cm. in diameter, which stands 1 cm. above the ovary There are four transparent areas, fluctuating, varying in size up to 1 cm. These are apparently developing follicles. On section, contains a corpus luteum, 1·8×1·7×1·4 cm., somewhat hourglass-shaped and orange-yellow in colour. Close to the corpus luteum is a Graafian follicle 1·5 cm. in diameter; two smaller Graafian follicles are also present. The remaining portion of the ovary is greyish-white in colour and firm, with several small blood-vessels.	*Thyroid gland.*—Normal. *Pancreas.*—Normal. *Epiphysis.*—Normal. *Hypophysis.*— (1) Pars distalis—normal. (2) Pars intermedia—increase in connective tissue, which shows numerous fat droplets. Local accumulation of lymphocytes. (3) Pars nervosa—normal. *Supramammary lymph gland.*—Small necrotic area surrounded by an area with eosinophilic infiltration. *Mammary gland.*—concretions, mammitis catarrhalis. *Cervix.*—Normal. *Uterus.*—Metritis chronica catarrhalis. *Fallopian tubes.*—pavilionitis, hydrosalpinx. *Ovary.*— Right—fibrous filaments, corpus luteum normal histologically. Left—cystic degeneration, cyst shows lutein tissue in wall. *Tumour.*—Fibrous capsule, conteuts show fibrin.	22.8.25: Corpus luteum expressed. Uterine irrigation with 1 per cent. Lugol's iodine and swabbing of the os with Lugol's iodine was carried out five times, also uterine massage. 29.10.25: Cyst expressed from right ovary. 12.2.26: corpus luteum expressed from right ovary. Salpingitis and pavilionitis developed during treatment. The case was then considered hopeless and slaughtered.	Corpus luteum persistens, pavilionitis, endometritis catarrhalis chronica. This was followed by hydrosalpinx and cystic degeneration of the corpus luteum.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·7 × ·8 × 16 cm.	Weight 0·42 gm.
Hypophysis, 2 × 1·6 × 1·5 cm.	„ 3·0 gms.
Adrenals—Right, 7 × 4 × 1·3 cm.	„ 20·0 „
Left, 6·2 × 5 × 1·3 cm.	„ 16·5 „

Thyroid—Right half, 4·8 × 6·4 × ·7 cm.
Left half, 6 × 6·3 × ·8 cm.
Length, including isthmus, 31·8 cm., weight 30 gms.

CASE 21. (See Appendix II, Fig. 21.)

FRIESLAND COW 1660, 11 YEARS OLD.

Date of first examination, 22.8.25. *Date of slaughter*, 11.8.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 945 lb. Normal in appearance. Udder is in the later stages of lactation. Its tissue is soft and pliable. While under observation for 12 months, oestral periods have occurred at regular intervals except that there was intermission in the month of July. The cow has had several calves, the exact number is unknown. Date of last parturition — June, 1923. Contagious abortion test—negative. Tuberculin test—positive.	The vulva is normal. Vagina contains a small quantity of clear mucus. Cervix: first cervical fold is ectropic and ragged, mucosa slight reddish. The ostium uterinum admits the index finger for an inch. From the rectum the cervix seems somewhat enlarged, but normal in consistency. The uterus and uterine horns are almost entirely abdominal. Consistency is soft and flabby. Both horns are somewhat larger than normal. Both fallopian tubes are normal in size and consistency. The right ovary is elongated, about 5×3 cm. It is soft in consistency, but no definite cyst formation can be felt. The left ovary is about equal in size to the right. It contains a corpus luteum. This ovary is also slightly soft, but no definite cystic formation can be dtermined. Broad ligaments are normal. *Diagnosis*: endometritis chronica catarrhalis and cervicitis. While under ovservation for twelve months some hard nodules developed in the right uterine horn. The condition remained otherwise unchanged.	Vulva is normal. The vagina shows some small abscesses, up to the size of a pea, along the course of the ducts of Gaertner. Cervix, 8 × 6 cm.; width of wall, 3·5 cm.; the first cervical fold is ectropic and ragged. mucosa pale-pink in colour. Uterus, 13 × 7 cm.; width of wall, 1·5-2 cm.; left horn, 20 × 4 cm.; width of wall, 1·5 cm.; right horn 22 × 5 cm.; width of wall, 1—1·5 cm. The mucosa is yellowish-pink to slaty-pink in colour; it is smooth, swollen, and oedematous. In parts it is transparent. On palpation the wall of the uterus is soft. The right horn contains several small nodules varying from a pin's head to a pea: these are almost entirely confined to the mucosa. On section, they are yellowish in colour and gritty. Some of the larger nodules extend to the muscular coat. The fallopian tubes are unchanged in size and consistency. The broad ligaments are normal. Post-mortem showed tuberculosis of the peritoneum, pleura, lungs, and bronchial lymphatic glands.	Left ovary, 5 × 3 × 2·5 cm., oval in shape. Surface smooth and glistening. It shows numerous small cyst-like structures on the surface, varying in size up to 1·5 cm. in diameter. Towards the anterior pole there is a large cyst-like structure. apparently a developing Graafian follicle, 1·7 cm. in diameter. On palpation the ovary is soft. On section. there are traces of several atrophied corpora. The cyst-like structures are apparently normal follicles. The ovarian tissue is greyish-white and moist. Right ovary, 5 × 3 × 2·5 cm., shows similar outwards appearance to the left, with numerous cyst-like prominences. Towards its anterior pole there is a prominent corpus luteum of the last interovulation period; five yellow specks, the remains of previous corpora lutea, are seen through the surface. On palpation the ovary is soft. Section shows a lemon-coloured corpus luteum 1·4 cm. in diameter. The cyst-like structures seen through the surfaces are apparently normal follicles. There are several traces of atrophied corpora lutea. The ovarian tissue is whitish-grey and moist.	*Adrenals.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Hypophysis.*—Normal. *Epiphysis.*—Corpora. amylacea. *Pancreas.*—Normal. *Mammary gland.*—slight catarrhal mammitis. *Lymph gland.*—Slight fibrosis. *Vagina.*—Abscess in duct of Gaertner (tubercular). *Cervix.*—Fibrosis. *Fallopian tubes.*—Normal. *Ovary.*— Right—normal follicles, corpus luteum with much fibrous tissue. Left—atretic follicle (follicular epithelum filling the cavity), small cyst with flattened epithelial lining and wall of connective tissue and theca cells. Normal folicles. *Uterus.*—Metritis chronica catarrhalis. (tubercular metritis).	The uterus was irrigated on several occasions with 1 per cent. Lugol's iodine and the cervix swabbed with pure Lugol's iodine. Uterine massage was also tried. Tubercular nodules developed in the right uterine horn during the time the cow was under observation.	Tubercular metritis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·4 × ·7 × ·7 cm. .... Weight 0·4 gm.
Hypophysis, 2·4 × 1·8 × 1·4 cm. .... ,, 3·5 gms.
Adrenals—Right, 5 × 3·6 × 1·6 cm. .... ,, 14·5 ,,
Left, 4·6 × 2·8 × 1·5 cm. .... ,, 11·0 ,,

Thyroid—Right half, 4 × 4·5 × 1 cm.
Left half, 5 × 2·6 × 9 cm.
Length, including isthmus, 21·2 cm., weight 20 gms.

CASE 22. (See Appendix II, Fig. 22.)

FRIESLAND COW 1662, 15 YEARS OLD.

Date of first examination, 27.8.25. *Date of slaughter*, 11.5.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition fair. Weight 1,170 lb. There is very slight relaxation of the pelvic ligaments: the appearance is otherwise normal. The cow has lactated, but is now dry. No. of calves, several. Last parturition—March, 1925. Periods of oestrum irregular. The animal has had contagious granular vaginitis. Contagious abortion test—negative. Tuberculin test—negative.	The vulva shows a few old nodules of granular vaginitis. The labiae show a few old cicatrices. The vagina contains a quantity of thick yellowish-green foetid pus. The posterior extremity of the cervix is markedly enlarged and the first two cervical folds are ectropic. The mucosa is dark red in colour. The os contains thick pus and admits the finger to its entire depth. Per rectum, the cervix is enlarged and firm. The uterus and uterine horns are normal in size, but slightly flaccid. Both fallopian tubes are normal, but there are adhesions between the fimbriated extremity and the ovary. Right ovary normal in size and consistency. Left ovary, size of a small hen egg, somewhat fluctuating. At its anterior extremity there is a prominent corpus luteum. Broad ligaments normal. *Diagnosis*: cervicitis purulenta and endometritis chronica. While under observation this cow developed pyometra, salpingitis, and cystic oophoritis. The mucosa of the cervix became normal in colour but there was little reduction in size.	The vulva shows some nodules of granular vaginitis. There are a few cicatrices on the labiae. The vagina contains a small quantity of thick greenish-yellow very foetid pus. The cervix is markedly ectropic. The posterior extremity protrudes in to the vagina as a large cauliflower-like growth. The first and second cervical folds are inverted and the third partly inverted. The mucosa is pale, but in parts is ulcerated. Diameter, 10 cm.; length, 9·5 cm. On section, width of wall posteriorly is 4·5 cm., anteriorly 3·5 cm. The os uterus is patent. The uterus and uterine horns are normal in size, but somewhat flaccid, soft, and thickened. Uterus body, 12·7 cm., wall 1·5 cm.; horns—right, 19·5 × 3·5 cm.; left, 20 × 3·5 cm.: wall, 1·5 cm. to 2 cm. The mucosa is greyish-yellow in colour, soft, and moist. It appears to be somewhat wsollen, up to ·5 cm. in depth. The cotyledons are fairly prominent, greyish-yellow in colour, giving the mucosa a mottled appearance. Both fallopian tubes are enlarged and firm, ·4 cm., except at the ovarian extremity, where they are slightly fluctuating. The left shows a cystic formation 4 cm. from the fimbriated extremity. Both extremities are loosely adherent to the ovary. The attachment is irregular, so that the extremity does not form an enclosed cavity. The broad ligaments are normal.	Right ovary, 4·8 × 4·2 × 3·5 cm. It is oval and covered by the fimbriated extremity of the fallopian tube to which it is irregularly adherent. It is fluctuating and appears cystic throughout. There are traces of five atrophied corpora lutea seen on its surface. On section it shows a large cystic corpus luteum which fills almost the entire ovary. Only a thin capsule of ovary tissue remains. Left ovary, 4 × 2 × 1·8 cm. It is somewhat elongated. There are numerous fibrous adhesions; these are in part stained brown, apparently the result of an old haemorrhage. The ovary is firm in consistency. There are two atrophied corpora lutea apparent on the surface. On section, the ovarian tissue appears unchanged. There are three atrophied corpora lutea and two apparently normal Graafian follicles present.	*Adrenal.*— Right—normal. Left—normal. *Thyroid.*—Normal. *Epiphysis.*—Few corpora amylacea. *Hypophysis.*—Pars distalis contains two cysts lined with epithelium, contents of colloid nature with epithelial cell débris. *Supramammary lumph gland.*—Shows neutrophilic infiltration and oedema. *Mammary gland.* numerous concretions. *Pancreas.*—Normal. *Fallopian tubes.*—Hydrosalpinx. *Uterus.*—Metritis chronica catarrhalis. Capillaries show some hyaline degeneration. *Cervix.*—Cervicitis chronica purulenta et ulcerosa. *Ovary.*— Right—lutein cyst, atretic follicles, oedema, fibrous attachments. Left—small cystic degeneration, atretic follicles, some oedema, fibrous filaments.	The cervix was swabbed with Lugol's iodine and the uterus irrigated with 1 per cent. Lugol's iodine on 10 occasions, between 27.8.25 and 1.1.26. When pyometra supervened, draining of the purulent contents of the uterus was followed by uterine irrigation and the insertion of an antiseptic pessary. An embedded corpus luteum was also expressed during treatment.	Cervicitis chronica purulenta, endometritis chronica catarrhalis followed by pyometra, salpingitis, and cystic degeneration of the corpus luteum.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·4 × ·5 × ·6 cm. .... Weight 0·3 gm.
Hypophysis, 2·5 × 1·7 × 1·6 cm. .... „ 3·0 gms.
Adrenals—Right, 5 × 3·7 × ·8 cm. ..... „ 12·0 „
Left, 4·5 × 4·4 × 1·1 cm. .... „ 13·0 „

Thyroid—Right half, 4·5 × 4·4 × ·8 cm.
Left half, 5 × 3·2 × ·6 cm.
Length, including isthmus, 22 cm., weight 14·5 gms.

CASE 23. (See Appendix II, Fig. 23.)

FRIESLAND COW No. 1663, 10 YEARS OLD.

Date of first examination, 27.8.25. *Date of slaughter*, 18.2.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition poor. Weight 1,160 lb. Normal in appearance. Has lactated, but is now dry. Udder tissue is normal. Number of calves—3. Date of last parturition—24.6.24. Since last parturition the oestral periods have been very irregular, with abnormally long inter-ovulation periods. The cow had not shown oestrum for four months prior to slaughter and had only one oestral period in ten months. Contagious abortion test—negative. Tuberculin test—negative. While under observation the pelvic ligaments became relaxed and the root of the tail became elevated. The cow never showed symptoms of abnormal sexual desire.	Vulva shows several old cicatrices and some warty growth on the skin. There is a slight mucous discharge. Several small yellow transparent nodules present in mucosa. Vagina, two small nodules the size of a pea along the right duct of Gaertner. Small quantity of tenacious mucus present. Cervix—first cervical fold ectropic and dark-red in colour, ostium uterinum admits one finger easily to a depth of 3 cm. From the rectum the cervix appears normal in size and slightly firm. Uterus and uterine horns are normal in size, but soft on palpation. Both fallopian tubes are normal. Right ovary is somewhat large, firm in consistency. Surface is smooth. Left ovary is also somewhat large, firm in consistency. Surface smooth. Broad ligaments—normal. *Diagnosis*: cervicitis, metritis chronica catarrhalis,	Vulvar mucosa normal. Vaginal mucosa normal. Cervix, 7×6 cm.; width of wall, 3 cm.; first cervical fold hypertrophic and ectropic, mucosa yellowish-pink in colour. The ostium uterinum contains some thick tenacious mucus. Uterus—body, 12 × 4 cm.; width of wall, 1 cm.; left horn, 17×3 cm.; width of wall, ·7 cm.; right horn, 16×3 cm.; width of wall, ·7 cm. Uterus and uterine horns soft on palpation. Serosa smooth, yellowish-pink in colour. On section, it contains a small quantity of clear tenacious mucus. Mucosa brownish-yellow in colour. The cotyledons are fairly prominent, also brownish-yellow in colour, except towards the apices, where there is no trace of cotyledons. In the latter portion of the mucosa, one recognizes several small cysts pinhead in size. Both fallopian tubes are normal. Broad ligaments normal.	Right ovary, 5·5 × 4×2·5 cm. Capsule yellowish in colour, softish on palpation. Its surface presents a number of small areas, irregular in outline, varying up to 1 cm. in diameter. These areas appear as blisters raised above the surface of the ovary; their capsule is clear, transparent. On close examination these areas are composed of a number of small cysts, clustered together, separated by a thin wall. They extend more or less deeply into the cortex of the ovary. On section, the ovarian tissue is pinkish-grey and firm in consistency. There are large cyst-like areas and several small cyst-like areas apparent on the surface of section. Left ovary, 6 × 4·3 × 2·8 cm. Soft on palpation. Capsule yellowish in colour. Shows on its surface some small cyst-like areas, apparently normal follicles. There are also a number of the small multilocular cysts as described on the right ovary, but fewer in number. On section, ovarian tissue greyish-pink and firm. Several cyst-like structures varying in size. Some irregular areas of lutein tissue are also present.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Adrenals.*—Normal. *Supramammary lymph glands.*—Fibrosis. *Mammary gland.*—Numerous concretions. *Vulva.*—Normal. *Vagina.*—Normal. *Cervix.*—Fibrosis. *Uterus.*—Metritis chronica catarrhalis cystica. *Fallopian tubes.*—normal mucous and muscular zones. There is perisalpingitis. *Ovaries.*—Several small single and multilocular cysts, lined with epithelium; atretic follicles. Graafian follicles showing degeneration of the zona granulosa. No ova seen in these follicles.	Uterus irrigated with Lugol's iodine 1 per cent., followed by uterine and ovarian massage. Treatment carried out with weekly intervals for a month. Massage continued for four months at weekly intervals. In April cysts developed in both ovaries; treatment was again commenced after rupturing the ovarian cysts. In May the cow showed oestrum for the second time and was served without result. In October oestrum again appeared; the cow was served without result. About this time the surface of the ovaries became granular to the feel, the granular areas increasing in numbers until she was destroyed. During the last three months of observation the ovaries were somewhat soft on palpation, but cyst formation was not definitely diagnosed.	Metritis chronica catarrhalis, with cervicitis; this was followed by cystic oophoritis and cystic degeneration of the uterine mucous glands.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·6 × 2 × 1·4 cm.	Weight	4·5 gms.
Epiphysis, 1·6 × ·5 × ·5 cm.	,,	0·6 gm.
Adrenals—Right, 4·7 × 4 × 1·4 cm.	,,	20·0 gms.
Left, 6·8 × 2·6 × 1·2 cm.	,,	17·5 ,,

Thyroid—Left half, 4·5 × 3·5 × 1 cm.
Right half, 5 × 3·2 × 1 cm.
Length, including isthmus, 22 cm., weight 18·5 gms.

CASE 24. (See Appendix II, Fig. 24.)

FRIESLAND COW 1664, 6 YEARS OLD.

Date of first examination, 27.8.25. *Date of slaughter*, 8.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,180 lb. Normal in appearance. Has lactated. Date of only parturition—August, 1923. No history of contagious granular vaginitis. While under observation the oestral periods were very irregular. Contagious abortion test—negative. Tuberculin test—negative.	The vulva and vagina are normal. The first cervical fold is slightly ectropic and somewhat ragged. It is pale in colour. The os is patent. It contains a small quantity of slightly turbid, tenacious mucus. Per rectum, the cervix is normal in size and consistency. The uterus is slightly flaccid and its body somewhat broader than normal. Both uterine walls are also flaccid and somewhat enlarged. The right fallopian tube is normal in size. The left fallopian tube is markedly thickened, especially towards its fimbriated extremity, which is adherent to the left ovary: it is very convoluted and is firm on pressure, due to tension with contained fluid. The right ovary contains a prominent corpus luteum from the last interovulation period. The left ovary is the size of a walnut and firm in consistency; it is adherent to the fimbriated extremity of the fallopian tube. The broad ligaments are normal. *Diagnosis*: metritis chronica catarrhalis, cervicitis catarrhalis, hydrosalpinx, corpus luteum persistens.	The vulva is normal. The vagina contains a small quantity of transparent tenacious mucus. Cervix—length, 6 cm.; width of wall, 3 cm.; mucosa pale. The os is patent. It seems normal in consistency. Uterus—body, 10 cm.; horns, 14 cm.; width of body 4 cm.; horns, 2·5 cm.; depth of wall varies from 1 to 1·2 cm.; the serosa is yellowish-pink in colour. The uterus is somewhat soft on palpation. On section, it contains a quantity of clear viscid mucus. The mucosa is pinkish-grey in colour; it is swollen, oedematous, and somewhat transparent. The cotyledons vary in size up to 1 cm.; they are greyish-yellow to pinkish-grey in colour. The left fallopian tube is enlarged and tortuous throughout its entire extent. It gradually increases in diameter towards its fimbriated extremity, which partly encapsules the ovary. On section, the tube contains clear fluid. The fimbriated extremity is adherent to the ovary over its free border. The right tube shows five small isolated cysts at its uterine extremity. The remainder of the tube appears normal. The cysts contain a clear fluid.	Right ovary, 4 × 3·6 × 2·5 cm. Its surface shows numerous follicles in various stages of development. The largest of these is 1·3 cm. in diameter. It shows the presence of seven atrophied corpora lutea on its surface: it also contains the corpus luteum of the last interovulation period. On section, there is a pale lemon-coloured corpus luteum of the last interovulation period, 1·5 × 1·4 cm. There are several Graafian follicles, varying in size from 3 mm. to 1·3 cm. in diameter. The remainder is greyish-pink in colour and appears normal in consistency. Left ovary, 3 × 2·1 × 2 cm. It is almost completely encapsuled by the fimbriated extremity of the fallopian tube through which its surface cannot be observed. It is firm in consistency. When the capsule of the fimbriated extremity is removed, the corpus luteum of the second last interovulation period appears on the surface. Slightly protruding above the surface, a few developing Graafian follicles can also be seen. On section, a pale lemon-coloured corpus luteum of the second last interovulation period is present, 1·4 × ·9 cm. There are several developing Graafian follicles varying in size up to ·5 cm. in diameter. The remainder of this ovary is pinkish-grey and normal in consistency.	*Epiphysis.*—Corpora amylacea. *Hypophysis.*—Normal. *Adrenals.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Sclerosis. *Vagina.*—Vaginitis catarrhalis chronica. *Uterus.*—Metritis catarrhalis chronica with commencing cystic formation. *Fallopian tubes.*—hydrosalpinx. *Ovaries.*— Right—normal follicles, corpus luteum (1) present, which is rich in stroma and poor in lutein cells. Left—fibrous adhesions on surface. Corpus luteum (2) present, poor in lutein cells and rich in stroma.	Case considered hopeless at first examination. No treatment tried. The animal was kept under observation for a period of 7½ months. During this time little change took place. A few small cysts developed at the uterine extremity of the right fallopian tube.	Metritis chronica catarrhalis cystica, cervicitis catarrhalis, and bilateral hydrosalpinx.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·8 × ·6 × ·6 cm.	Weight 0·45 gm.
Hypophysis, 2·2 × 1·5 × 1·3 cm.	„ 2·5 gms
Adrenals—Right, 6·1 × 3·6 × 1·3 cm.	„ 17·5 „
Left, 5·1 × 4·4 × 1·5 cm.	„ 16·0 „

Thyroid—Right half, 4·8 × 3·7 × 1·2 cm.
Left half, 3·8 × 4·7 × 1 cm.
Length, including isthmus, 22·2 cm, weight 22·5 gms.

CASE 25. (See Appendix II, Fig 25.)

FRIESLAND COW 1665, 10 YEARS OLD.

Date of first examination, 27.8.25. *Date of slaughter*, 17.8.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Normal in appearance. Weight 1,160 lb. Number of calves—1. Date of parturition —March, 1920. Contagious abortion test —negative. Tuberculin test—negative While under observation at this Institution the cow showed nine oestral periods in two years: on two occasions six months passed without any evidence of oestrum.	Vulva normal. Vagina contains a small quantity of tenacious slightly opaque mucus. Cervix: the pars vagina is somewhat enlarged and slightly ragged; ostium uterinum is patent for a small dilator. Uterus is normal in size and of fairly good tone. The fallopian tubes are normal. Right ovary is the size of a walnut, firm; contains the corpus luteum of the last interovulation period. Left ovary is the size of a walnut, firm; there is a small crater at its anterior extremity, the result of a recently ruptured Graafian follicle. Broad ligaments normal. *Diagnosis*: No palpable change in the genitalia.	Vulva normal. Vagina normal. Cervix, 10 × 4 cm.; width of wall, 2 cm.; mucosa pale. There is a small false cervical plug present. Uterus—body, 7 × 5 cm.; width of wall, 1·2 cm.; right and left horns, 20 × 3 cm.; width of wall, ·8 cm.; serosa normal. The cavum uteri contains 50 c.c. of opaque slimy mucus which is chiefly confined to the right horn. Mucosa of the left horn is pale in colour and somewhat moist; the cotyledons are atrophied. The mucosa of the right horn is greyish-red in colour and moist; the cotyledons are slightly swollen and reddish in colour. Both fallopian tubes are unchanged. The broad ligaments are unchanged.	Right ovary, 4 × 2·5 × 2·5 cm.; surface smooth; it contains the corpus luteum of the last interovulation period, which had up to the time of slaughter existed for four months. On section, there is a brownish-orange corpus luteum, 1·5 cm. in diameter. Some small cyst-like structures (follicles) are also present. The ovarian tissue is pinkish-white and firm in consistency. Left ovary, 3 × 2·4 × 2 cm.; surface smooth; underneath the surface some small cyst-like areas are present, over which the capsule is thin and transparent (follicles). On section, there are several small cyst-like structures, apparently normal follicles. The ovarian tissue is greyish-white to greyish-pink in colour and firm in consistency.	*Epiphysis*.—Normal. *Hypophysis*.—Normal. *Adrenals*.—Slight irregularity in the distribution of lipoid granules in the cortex. *Thyroid*.—Normal. *Thymus*.—Normal. *Pancreas*.—Normal. *Supramammary lymph gland*.—Normal. *Mammary gland*.—Normal. *Vagina*.—Normal. *Cervix*.—Normal. *Fallopian tubes*.—Mucous folds appear thickened with fibrous tissue. *Ovaries*.—Small cysts with epithelial lining. Normal follicles present. Normal corpus luteum. *Uterus*.—Metritis chronica catarrhalis, with atrophy of the mucosa and diminution in uterine glands.	27.8.25–28.10.25: Uterine and ovarian massage. 12.2.26: Corpus luteum persistens expressed. 7.4.26: Uterus irrigated 1 per cent. Lugol's iodine, cervix swabbed Lugol's iodine. 7.4.26–7.5.26–7.6.26: 1½ c.c. pituitary infundibular extract, 1 c.c. adrenalin intravenously.	Metritis chronica catarrhalis, with atrophy of the mucosa and diminution in uterine glands.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 2 × ·75 × ·75 cm..........	Weight 0·75 gm.	Parathyroid, 1 × ·75 × ·3 cm.
Hypophysis, 2·5 × 2 × 1·25 cm.........	,, 3·0 gm.	Thyroid—Left half, 11 × 8 × ·75 cm.
Adrenals—Left, 4·5 × 4·25 × 1·5 cm....	,, 16·0 gms.	Right, 7 × 6 × ·75 cm.
Right, 5·2 × 3·2 × 1·25 cm..	,, 14·5 gms.	Length, including isthmus, 23·5 gms., weight 36 gms.

CASE 26. (See Appendix II, Fig. 27.)

FRIESLAND COW 1682, 12 YEARS OLD.

Date of first examination, 28.8.25. *Date of slaughter*, 21.11.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,050 lb. The animal shows signs of old age. Has lactated. The right posterior quarter of the udder is swollen, firm on palpation, and contains a large quantity of thick greenish-yellow purulent material. The tail and hind-quarters are stained with a yellowish discharge which issues from the vulva. No of calves—unknown. Last parturition—March, 1925. Since last parturition, oestrum had not been carefully observed. The cow had been bred several times, but failed to conceive. While under observation at this Institution she never showed oestrum. Contagious abortion test—negative. Tuberculin test—negative.	Vulva shows dormant lesions of granular vaginitis. The vagina contains a quantity of tenacious muco-purulent material which is somewhat evil-smelling. The mucosa of the vagina is pale. The posterior extremity of the cervix is markedly enlarged and ragged; the first fold is altogether ectropic and the second fold is partly protruding. The mucous membrane is red in colour. The cervix is abnormally firm. The cervical canal admits the finger to its entire depth. The uterus and uterine horns are enlarged and flaccid, the walls thin, atonic, and there is slight fluctuation. The right fallopian tube appears unchanged; the left is enlarged throughout, especially towards its fimriated extremity, where fluctuation can be detected. The right and left ovaries are firm in consistency and normal in size. There are adhesions between the ovaries and the mesosalpinx. The broad ligaments are somewhat relaxed. *Diagnosis*: cervicitis, pyometra, salpingitis. During treatment this cow developed peritonitis, with adhesions between the rectum, uterus, broad ligaments, ovary and bladder.	The vulva shows some lesions of granular vaginitis. The vagina contains a small quantity of purulent material. The cervix, 10 × 8 cm.; is enormously enlarged. The posterior opening is irregularly ectropic, its mucosa is reddish in colour. The uterine body and horns are enlarged; the serous surface is irregularly covered with fibrous adhesions. It is attached to the rectum and to the ileum. On the dorsal wall of the uterus immediately cranial to the cervix is a firm swelling, the size of an ostrich egg, which contains a creamy purulent material, with some whitish yellow flocculi. The cavity communicates with the uterus by a small opening 3 or 4 mm. in diameter; the opening is situated 1·5 cm. from the cervix. On section, the uterine wall is thin, ·8 to 1·5 cm.; where it is not adherent to the pelvic organs its mucosa is diffusely reddened. The cotyledons are yellowish-white in colour, varying in size from ·5 to 1·3 cm. in diameter. The right fallopian tube is enlarged; its fimbriated extremity is adherent and cystic; it is difficult to trace the tube on account of fibrous adhesions. The left fallopian tube is impossible to trace, as it is covered in a dense mass of newly formed tissue. Broad ligaments appear lengthened. The udder is enlarged, especially the right hindquarter, which is firm. On section, there are some caverns which are filled with a dirty, greenish purulent material.	Right ovary, 4 × 2½ × 2 cm.; is surrounded by fibrous material, the result of fibrous peritonitis; its fallopian tube is adherent. On section, there is a cyst-like structure, 1 × ·6 cm., which contains a clear yellow fluid. There are two corpora lutea present, (1) 1·3 × 1 cm. (2) 1 × ·9 cm.; one is central, while the other projects. There is an atrophied corpus luteum present close to the deeper wall of the cyst above described; the remaining portion of the ovary is greyish-white and fibrous looking. Left ovary, 2·8 × 1·8 × 1·8 cm., is fibrous on palpation: it is surrounded by fibrous adhesions. On section, it shows the presence of three atrophied corpora lutea. The remaining part of the ovary is greyish-white in colour. There is no evidence of developing Graafian follicles.	*Cervix.*—Cervicitis catarrhalis. *Uterus.*—Pyometra, abscess uterine wall. *Fallopian tubes.*—pavilionitis, salpingitis. *Ovaries.*— Left—fibrous tissue adhesions to surface. Right—fibrous tissue adhesions to the surface, corpus luteum appears unchanged, small cyst (Graafian follicle type). *Mammary gland.*—Induration and local abscess. *Endocrine glands.*—Not examined histologically.	The uterus was irrigated with 1 per cent. Lugol's iodine, and the cervix swabbed with Lugol's iodine weekly for five weeks. During treatment the cow developed local peritonitis, involving the structures in the pelvis, and an abscess in the dorsal wall of the uterus, probably as a result of injury during treatment.	Cervicitis, pyometra salpingitis, and corpus luteum persistens.

CASE 27. (See Appendix II, Fig. 28.)

FRIESLAND COW 1683, 5 YEARS OLD.

Date of first examination, 28.8.25. *Date of slaughter*, 5.11.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition very poor. Weight 822 lb. Normal in appearance. Has lactated, but the udder is now non-lactating, soft, and pliable. No. of calves. 1. Date of parturition 1924. The cow suffered from retained afterbirth and metritis and had never regained normal health. She had not shown oestrum since calving. Contagious abortion test, positive. Tuberculin test, positive.	Vulva does not admit the hand. There are numerous nodules of contagious granular vaginitis present. When examined from the rectum, the vulva and vagina show nothing unusual. The cervix appears normal, but in close proximity to the cervix, in the pelvis, there is a swelling the size of an orange, which is firm. The uterus cannot be accurately palpated owing to the presence of adhesions between it, the bladder, and the broad ligaments. Its body and horns appear normal in size, but there are numerous fibrous adhesions to the serous surface of the horns, making them difficult to examine. The fallopian tubes cannot be examined owing to fibrous adhesions. The right ovary is involved in a mass of newly formed tissue and cannot be definitely palpated. The left ovary is the size of a pigeon's egg: it appears normal in consistency. The right broad ligament contains two firm swellings, one the size of an orange, the other somewhat smaller. The left broad ligament contains a similar swelling about 6 cm. in diameter. *Diagnosis :* perimetritis, parametritis, perisalpingitis, pavilionitis (probably tubercular).	The vulva shows some greyish-yellow nodules. The mucosa of the vagina shows no change. The external opening of the cervical canal shows a slightly ectropic posterior fold. The mucosa of the cervical canal is normal; it is 6·5 cm. long. The uterus: body, 7 cm. long; right horn, 16 cm.; left horn, 19 cm.; the serous surface of the uterus is covered with adhesions of a fibrous nature; the horns are similarly involved. On section, the uterus shows an apparently normal mucosa; its wall also appears normal. Fallopian tubes—right, about its centre is much convoluted and shows a mass of small cysts 3 cm. in diameter, its fimbriated extremity is almost entirely adherent to the ovary; left, towards its centre shows a convoluted mass of cysts 2·5 × 1·5 cm.; its fimbriated extremity is adherent to the ovary; it is markedly enlarged, its wall thickened and covered with fibrous filaments. It contains a somewhat opaque fluid. The broad ligaments contains three rounded tumors varying in size up to an orange. On section, these have a firm fibrous capsule and a yellow slightly caseous centre. Tubercular lesions in the bronchial lymphatics, mediastinal lymphatics, and peritoneum.	The right ovary is 4 × 2½ × 1½ cm. It presents some fibrous filaments on its surface. There are two atrophied corpora lutea apparent on the surface. On palpation it is somewhat fluctuating. The fimbriated extremity of the fallopian tube is attached by fibrous adhesions. On section, the ovary presents four cyst-like structures varying in size from ·6–1·2 cm. in diameter. These cysts contain slightly yellowish fluid. There is also an atrophied corpus luteum present. The remaining portion of the ovary has a greyish-white fibrous appearance. The left ovary 3 × 2 × 2 cm. It is rounded; its surface is adherent by fibrous filaments to the swollen extremity of the fallopian tubes. There are also present some isolated fibrous filaments. On section, there is a large corpus luteum present, 1·6 cm. diameter. It is pale yellow in colour and shows at its centre a cyst ·5 cm. in diameter. The cyst contains a clear yellowish fluid. There is also a smaller atrophied corpus luteum present. The remainder of the ovary is greyish-white in colour and firm in consistency.	*Epiphysis.*—Numerous corpora amylacea *Hypophysis.*—Four vesicles in pars distalis, lined with cubical epithelium. *Adrenal gland.*—Right and left, Normal. *Supramammary lymph gland.*—Hyperaemia. *Mammary gland.*—Concretions. *Thyroid gland.*—Stroma colloides. *Vagina.*—Normal. *Cervix.*—Normal. *Ovary.*— Right, fibrous filaments. Left, fibrous filaments, cystic degeneration of the corpus luteum. *Fallopian Tubes.*— Right, hydrosalpinx. Left, hydrosalpinx. *Uterus.*—Perimetritis, parametritis.	Prognosis hopeless. No treatment tried.	Corpus luteum, which is undergoing cystic degeneration, hydrosalpinx, perisalpingitis perimetritis, parametritic abscess.

SIZE AND WEIGHTS OF ENDOCRINES.

Adrenals—Right, 5·4 × 3·75 × 1·25 cm. Weight 14·0 gms.
Left, 4·75 × 4·25 × 1·25 cm. „ 12·75 „

Thyroid—Right half, 4·5 × 5 × ·75 cm.
Left half, 5·5 × 4·5 × ·75 cm.
Length, including isthmus, 21 cm., weight 22 gms.

CASE 28. (See Appendix II, Fig. 29.)

FRIESLAND COW 1685, 11 YEARS OLD.

Date of first examination, 28.8.25. *Date of slaughter*, 5.5.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 790 lb. Cow lacks in constitution. Normal in appearance. Has lactated but is now dry. No. of calves, unknown. Date of last parturition, April, 1924. Oestral periods very irregular and infrequent. Cow has suffered from contagious granular vaginitis. Contagious abortion test, negative. Tuberculin test, positive.	Vulva shows some dormant nodules of granular vaginitis. Vagina is normal. The cervix is ragged. The first cervical fold is protruding, and the second cervical fold is plainly seen through the ostium uterinum externum. The mucosa is dark red in colour. A small quantity of tough tenacious material is present in the os uteris. The cervix is normal in length, but appears somewhat enlarged in circumference when examined from the rectum. The uterus is mostly abdominal. It is flaccid and lacking in tone. The uterine horns are flaccid. The right is longer. The fallopian tubes appear normal in size. There are adhesions between the fimbriated extremity and the ovary on both sides. Right ovary, normal in size, firm. Neither Graafian follicle nor corpus luteum can be palpated. Left ovary normal in size. It contains a prominent corpus luteum. Broad ligaments are normal. *Diagnosis*: cervicitis and adhesive pavilionitis.	The vulva shows some old standing lesions of contagious granular vaginitis. The vagina contains a small quantity of clear tenacious mucus. The cervix—length, 8 cm.; width of wall in posterior part 3 cm., and anterior part 2·3 cm. The pars vagina is ragged. The first cervical fold is protruding. The mucosa is pale, but shows red spots, some of which are confluent. The uterus—body, 16 × 6 cm.; horns—left, 16 × 3 cm.; right, 19 × 3 cm.; width of wall, 1·5 cm. The ventral serosa is adherent to the fundus of the bladder by loose filamentous adhesions. The serosa is reddish in colour, with a slight yellowish tinge. The wall of the uterus and horns is somewhat flaccid. The left fallopian tube is firm and somewhat thickened. Its extremity is attached to the ovary by fibrous adhesions; it forms a soft cyst. The right tube is firm and somewhat thickened; its extremity is attached to the ovary. Tubercular lesions were present in the bronchial and mediastinal glands.	Left ovary, 2·8 × 2·7 × 1·8 cm. Its surface shows the presence of some fibrous adhesions and fibrous filaments. It is firm in consistency and oval in outline. On section, there is a developing Graafian follicle 1·3 cm. in diameter. In the centre of the ovary there is a corpus albicans ·5 cm. in diameter. The remainder of the ovary is greyish-white and fibrous in nature. Right ovary, 4 × 3 × 2·1 cm. Its surface shows the presence of fibrous filaments and fibrous adhesions. It is oval in outline and firm in consistency. On section, it contains a large orange yellow corpus luteum 2·8 cm. in diameter. The centre of the corpus luteum shows a cyst-like cavity ·5 cm. in diameter. The lutein tissue is separated by strands of greyish connective tissue, which are very apparent. The corpus luteum is central and surrounded by a capsule of greyish-white ovarian tissue. There is also an atrophied corpus luteum present ·3 cm. in diameter, brownish in colour.	*Epiphysis.*—Corpora amylacea. Arterio-sclerosis in a few vessels. Small haemorrhage. *Hypophysis.*—Pars distalis, pars intermedia, pars nervosa-arterio-sclerosis in vessel wall. *Thyroid.*—Normal. *Pancreas.*—Normal. *Adrenals.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Cervix.*—Fibrosis. *Uterus.*—Perimetritis, slight chronic changes in mucosa. *Fallopian tubes.*—Hydrosalpinx. *Ovary.*— Right.—Normal follicles, corpus luteum appears normal. Left.—Normal follicles. Two small calcified nodules in connection with an atretic follicle.	28/8/25.—Cervix swabbed with Lugol's iodine, uterus douched with 1 per cent. Lugol's iodine. This treatment was continued at fortnightly intervals for six weeks. Afterwards the uterus was massaged weekly until 15/1/26, when examination showed that pavilionitis with cystic formation had developed. The cow was kept under observation until slaughtered.	Cervicitis followed by pavilionitis. Histological examination showed chronic changes in the uterine mucosa.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·4 × ·7 × ·7 cm. .... Weight 0·3 gm.
Hypophysis, 2 × 2·5 × 1·4 cm. .... ,, 3·5 gms.
Adrenals—Right, 5·5 × 4·2 × 2 cm. ..... ,, 19·5 ,,
Left, 5 × 4·8 × 1·8 cm. ...... ,, 15·0 ,,

Thyroid—Right half, 4·5 × 5 × ·8 cm.
Left half, 4·8 × 4·6 × ·8 cm.
Length, including isthmus, 23·5 cm., weight 23 gms.

CASE 29. (See Appendix II, Fig. 26.)

FRIESLAND COW 1686, 8 YEARS OLD.

Date of first examination, 28.8.25. *Date of slaughter*, 29.4.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility
		Genital Tract.	Ovaries.			
Condition good. Weight 1,130 lb. Normal in appearance. Has lactated, but the udder is now dry. Its tissue appears normal. No. of calves, unknown. Last parturition, 1924. Periods of oestrum very irregular and in frequent. Three oestral periods in six months. The cow has suffered from contagious granular vaginitis. Contagious abortion test, negative. Tuberculin test, positive.	The vulva shows numerous old lesions of granular vaginitis. The vagina is normal. There is a small hard nodule on its floor, the size of a pea, which is covered with mucosa. The cervix is normal in colour and shows a slightly protruding first fold. The os uterus admits a small dilator without difficulty. The uterus is mostly abdominal; its body is slightly flaccid and lacking in tone. The right uterine horn is somewhat larger than the left, both are flaccid. The right fallopian tube appears normal in size; its fimbriated extremity is attached to the ovary. The left fallopian tube is enlarged and fluctuating towards its anterior extremity. Its pavilion is attached to the ovary and is cystic. The right ovary is the size of a small hen egg, it is fluctuating and cystic. The left ovary is soft and fluctuating, little firm tissue can be felt in it. The broad ligaments are normal. *Diagnosis*: cystic ovary, salpingitis, and endometritis chronica catarrhalis. While under observation, both fallopian tubes became more enlarged, especially the right. Both fimbriated extremities became very much increased in size and fluctuating. The ovaries changed but little.	The vulva shows numerous lesions of granular vaginitis. The vagina is normal. The cervix shows a slight protruding first fold; its mucosa is pale with some small red spots. Length of cervix, 9 cm.; width of wall, ·9 cm. It is normal in consistency. The uterus 10 × 6 cm.; width of wall, 2 c.m.; left horn, 19 × 4 cm.; right horn, 18 × 4·3 cm. The cervix is of a yellow-red colour. Cotyledons are atrophied and represented by greyish, yellow, circular or oval depressions up to ·5 cm. in diameter. The right fallopian tube is very much dilated, reaching 2 cm. in diameter; its posterior two-thirds is fluctuating and contains a clear watery fluid: its anterior third is enlarged to 2·5 cm. in diameter; it is also fluctuating and reddish-blue in colour: it contains a reddish watery fluid; its fimbriated extremity is adherent to the ovary and is enormously enlarged, 7 × 6·2 × 5 cm.; it is bluish-grey in colour and fluctuating. On section, it contains watery blood-stained fluid. The anterior half of the left fallopian tube is dilated and fluctuating and contains a clear watery fluid. Its fimbriated extremity is adherent to the ovary, and is much enlarged, 5·8 × 3 × 4·2 cm. It is thin-walled and contains a clear watery fluid, which is slightly viscid. The cornual extremity of the tube is normal in size. There was a small tubercular lesion in the bronchial glands.	The right ovary, 5 × 3·5 × 3 cm., is embedded in a pocket in the broad ligament and is covered with connective tissue, through which it appears bluish-grey in colour; it is fluctuating and cystic. On section, the ovary contains two cysts, 2·8 cm. and 1·8 cm. in diameter. The cysts are divided by a thin septum. They contain a brownish watery fluid. The walls of the cysts are smooth, reddish-grey in colour. There is little ovarian tissue present. It has undergone atrophy. The left ovary, 4·5 × 3·5 × 3·5 cm., is partly adherent to the mesosalpinx; so that the fallopian tube lies attached to the medial aspect of the ovary; it is fluctuating throughout and is cystic; most of its capsule is transparent. Towards its anterior pole a corpus luteum is present, which projects somewhat above the surface. On section, there is a large cyst 3 cm. in diameter, which contains a clear yellowish slightly viscid fluid. The cyst wall is smooth. Its capsule is for the most part whitish and shows some yellowish lutein tissue in places. There is a corpus luteum, 2·5 × 1·8 cm., also present. This is orange-yellow in colour and contains a small cyst ·6 cm. towards its periphery. Situated in the small amount of ovarian tissue which remains there is a corpus albicans ·8 cm. in diameter.	*Hypophysis.*—Normal. *Epiphysis.* Slight increase in connective tissue. Three vesicles lined with columnar epithelium (1) 1350 × 675 *m*, (2) 1582 × 386 *m*, (3) 450 × 300 *m*. *Supramammary lymph gland.*—Induration and infiltration of eosinophiles. *Mammary gland.*—Normal. *Thyroid gland.*—Normal. *Thymus gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Induration. *Uterus.*—Metritis chronica catarrhalis cystica. *Fallopian tubes.*—Hydrosalpinx and pavilionitis. *Ovaries.*— Right.—Cystic degeneration (cysts wall of fibrous tissue), no normal follicles seen. Left.—Cystic degeneration of the corpus luteum. (1) Cyst wall of fibrous tissue and lutein tissue. (2) Cyst wall of lutein tissue with a thin inner lining of fibroblastic tissue and fibroblasts.	Case considered hopeless. No treatment carried out. The animal was kept under observation for a period of six months.	Endometritis, chronica catarrhalis, pavilionitis, cystic oophoritis, hydrosalpinx.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·5 × ·6 × ·6 cm.	Weight 0·3 gm.
Hypophysis, 2 × 1·6 × 1 cm.	„ 2·0 gms.
Adrenals—Right, 5·8 × 4·6 × 1·6 cm.	„ 14·5 gms.
—Left, 4·8 × 4·4 × 1·5 cm.	„ 14·0 gms.

Thyroid—Right half, 4·5 × 4·4 × 1·2 cm.
Left half, 6 × 3·5 × ·9 cm.
Length, including isthmus, 23·5 cm., weight 24·5 gms.

CASE 30. (Genitalia not photographed.)

AYRSHIRE COW 1805, 8 YEARS OLD.

Date of first examination, 23.10.25. *Date of slaughter*, 23.12.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition fair. There is a relaxation of the pelvic ligaments, which gives the rump a hollow appearance on either side. The root of the tail is elevated, and on either side there is a marked depression. The tail hangs slightly towards the left. The head has a somewhat masculine appearance. The cow has lactated. The udder is soft and pliable. Number of calves — 3. Last parturition — 1923. Since last parturition periods of oestrum have been irregular. Contagious abortion test—positive. Tuberculin test—positive.	Vulva shows a number of dormant nodules of contagious granular vaginitis. On the right wall there is a cyst the size of a pigeon's egg. The vagina contains thick tenacious mucus, with some white flocculi. The cervix shows ectropium of the first cervical fold. Its mucosa is of a dull reddish colour. The cervix, from the rectum, appears normal in size and consistency. The uterus is enlarged and is flaccid. The walls are somewhat thickened and appear soft. No fluid can be detected. Fallopian tubes are normal in size. Right ovary size of a walnut. It appears slightly soft in consistency. Left ovary size of a pigeon's egg. It also appears soft in consistency, as if it contains a central cyst. *Diagnosis*: cyst in duct of Bartholini, tubercular metritis, cystic ovaries.	Vulva shows a few small yellowish semi-transparant nodules. Vagina shows two small cystic prominences on its floor, 6 mm. in diameter. These contain clear liquid. Cervix, 9 × 5 cm., shows ectropium of the first cervical fold. Mucosa normal in colour. It appears normal in consistency. Uterus—body, 15 cm.; horns, 14 cm. in length. Its mucosa is of a brownish - yellow colour. It is covered with a slight yellowish-brown exudate. Scattered throughout the mucosa small yellowish spots varying in size up to a millet seed can be seen. On section, these areas can be seen dotted throughout the wall of the uterus. They are caseous on section, some are gritty. Fallopian tubes are not changed. P.-m showed tubercular lesions in the peritoneum.	Left ovary, 2·4 × 1·5 × 1·75 cm.; oval in shape, smooth on the surface. Slightly fluctuating. On section, there are three central cyst-like structures, and several atrophied *corpora lutea*. There is no evidence of ripening follicles. Right ovary, 3·5 × 1·5 × 1·7 cm., smooth on surface and slightly fluctuating. On section it presents two small cysts situated close to the pole further from the uterine horn. It shows the presence of many atrophied *corpora lutea*. There is no evidence of ripening follicles.	*Vagina*.—Mucosa normal, cysts in Gaertner's ducts. *Cervix*.—Sclerosis. *Uterus*—Metritis tuberculosa. *Fallopian tubes*.— Right normal. Left—slight thickening of the villi with connective tissue. *Ovaries*.—Small central cysts of the Graafian follicle type.	No treatment.	Tubercular metritis.

CASE 31. (See Appendix II, Fig. 30.)

AYRSHIRE COW 1806, 10 YEARS OLD.

Date of first examination, 23.10.25. *Date of slaughter*, 5.11.25.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Weight 948 lb. Condition fair. Head coarse. Neck slightly coarse. The root of the tail is slightly elevated. The pelvic ligaments are relaxed. The tail hangs towards the right side. Cow has lactated, but is now dry. There is abnormal sexual desire. Number of calves—2. ate of last parturition—1923. Both calves were weak at birth. Contagious abortion test—positive. Tuberculin test—positive.	The vulvar mucosa shows a few small dormant nodules of granular vaginitis. The vagina contains a small quantity of thick tenacious mucus with small white flocculi. The first cervical fold is ectropic and ragged. The os admits the finger to its full depth. It is normal in colour. The cervix is normal in circumference, but is very firm, almost hard as cartilage. The uterus is mostly abdominal. It is flaccid, lacking in tone. Its walls are soft and appear thickened. The uterine horns are also flaccid and have slightly thickened walls. The left fallopian tube is thickened and cystic throughout its entire length. The cystic condition is most marked towards the pavilion, which is adherent. The right fallopian tube is also markedly cystic anteriorly. The pavilion contains a cyst about 4 cm. in diameter. It is adherent to the ovary. Both ovaries are enlarged and cystic. The broad ligaments are somewhat relaxed. *Diagnosis* : cervicitis, metritis chronica catarrhalis, hydrosalpinx, cystic ovaries.	The vulva shows some yellowish semi-transparent nodules. Mucosa normal. The vagina shows the presence of some tenacious mucus mixed with whitish-yellow flocculi. Cervix. 8 cm. Anterior extremity very firm and fibrous. The posterior portion of the os is dilated and its first fold ectropic. Its mucosa is pale in colour. The uterus is 10 cm. long, 8 c.c wide. Horns — left, 19½ cm. : right, 23½ cm. There are some fibrous adhesions on its surface. The mucosa is slightly brownish in colour. There is a degeneration of the cotyledons, which are whitish-yellow in colour. Left fallopian tube, 14 cm., is enlarged, convoluted, bosselated, and cystic throughout its length. This is especially marked towards its ovarian extremity. The cysts vary in size up to 1 cm. The walls of the tube are thin. On section, a clear fluid flows away and the cystic parts collapse. The fimbriated extremity is adherent. The right fallopian tube 14 cm. long. It is also cystic, but the cystic portion is more marked towards its ovarian extremity. The posterior portion appears unchanged. The cysts are the same as those described in the left tube. The fimbriated extremity is attached to the ovary, so that the pavilion forms a large cyst 3½ cm. in diameter. P.-m. showed tubercular lesion in the bronchial lymph glands.	Left ovary, 6×4×3 c.m. It is lobulated and somewhat triangular in appearance. It is smooth on its surface and fluctuating. On section, it contains two large cysts 3 cm. and 1·5 cm. in diameter. These cysts contain a clear, slightly yellowish fluid. Each is covered with a thin fibrous capsule. There is a small triangular area of ovarian tissue between the cysts, which have caused atrophy of the ovary. Left ovary, 6×4×3½ cm. It has a lobulated appearance on account of cystic degeneration. Its surface is smooth. On section, it contains three large cysts and a smaller cyst from 1 cm. to 3 cm. in diameter ; each cyst is surrounded by a thin capsule of fibrous tissue. These cysts have caused atrophy of the normal ovarian tissue, so that only small portions remain.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Thyroid gland.*—Normal. *Supramammary lymph gland*—Normal. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Fibrosis. *Uterus.*— Metritis chronica catarrhalis cystica. *Fallopian tubes.*—Hydrosalpinx. *Ovaries.*—Cysts with connective tissue wall. No follicles seen. No lutein tissue present.	Case considered hopeless. No treatment.	Cystic oophoritis and hydrosalpinx, metritis chronica catarrhalis.

SIZE AND WEIGHTS OF ENDOCRINES.

Adrenals—Right, 5·75 × 3·75 × 1·5 cm.	Weight 17·5 gms.	Thyroid—Right half, 5 × 5 × ·6 cm.
Left, 4·5 × 4 × 1·25 cm......	„ 16·0 „	Left half, 5 × 4·25 × ·6 cm.
		Length, including isthmus, 20·5 cm., weight 20 gms.

CASE 32. (See Appendix II, Fig. 31.)

AYRSHIRE COW 1807, 12 YEARS OLD.

Date of first examination, 23.10.25. *Date of slaughter*, 2.3.26.

General Appearance and History	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 991 ℔. The root of the tail is elevated and the pelvic ligaments on both sides are relaxed. The head is somewhat coarse and ox-like The animal has lactated, but is now dry. Number of calves — 5. Last parturition — 1922 (calf dead and after-birth retained). Since then periods of oestrum have been irregular. Tuberculin test—Positive. Contagious abortion test —negative.	The lips of the vulva are covered with wart-like growths. The vulvar mucosa shows a few old nodules of granular vaginitis. Vagina contains small quantity of tenacious mucus. Cervix normal in shape and size. The mucosa of the cervix is slightly red in colour. The uterus is almost entirely abdominal it is normal in size, but is flaccid. The horns, especially towards the apices, are irregularly enlarged and the wall appears to be cystic, the enlarged areas being fluctuating and thin-walled. The right and left fallopian tubes appear thickened and firm. The right ovary is the size of a small hen egg; it is oval in shape and fluctuating. The left ovary is somewhat smaller: it is firm in consistency at its surface, but its centre is soft. It appears to contain a small central cyst. The broad ligaments are normal. *Diagnosis*: cystic oophoritis, cystic degeneration of the uterine wall, salpingitis.	The mucosa of the vulva shows a few small yellowish nodules. The posterior opening of the cervix shows nothing unusual. On section, it is 8 cm. long, firm, and its anterior half shows a number of small cysts up to 2mm. in diameter; these contain a transparent fluid. The uterine body is 9 cm. long, horns 25 cm. long. Through the serosa appear a number of small cyst-like elevations, which are greyish in colour. The apices of each horn are almost entirely cystic for a distance of 10 to 12 cm. The largest cyst is 3 cm. in diameter. On section the cysts contain a clear fluid and are very thin-walled. The uterine wall is thickened and soft; its mucosa presents a greyish-brown appearance; it is studded with cysts varying in size from 1 mm. to 2 cm.: some of these cysts extend deeply into the wall. The larger ones extend through the wall. The cotyledons have degenerated and are represented by small yellow areas 2 or 3 mm. in diameter. Both fallopian tubes are slightly thickened. The left contains a small cyst, 4 mm. in diameter. The fimbriated extremity of the left tube shows a slight fibrous attachment to the ovary. P.-m. showed tubercular lesion in the bronchial lymph glands.	Left ovary, 4 × 3 × 2·5 cm. Its surface presents a small fibrous adhesion; outwardly it is firm, but on pressure it is softish. On section, it contains a cyst-like structure 2 cm. in diameter, and a second one ½ cm. in diameter, apparently a normal follicle. The remainder of the ovary is fibrous and whitish in colour: there is no evidence of atrophied corpora lutea. Right ovary, 5 × 3 × 3·8 cm. It is oval and smooth on the surface, markedly fluctuating. On section it presents a large cyst and two smaller cyst-like structures surrounded by a thin capsule, 4–6 mm. The capsule is fibrous. The large cyst almost fills up the entire capsule. The two smaller cysts are 1 × 1·5 cm. in diameter.	*Epiphysis.*—Normal. *Hypophysis.* — Vascular engorgement. *Thyroid.*—Vascular engorgement. *Adrenals.*—Normal. *Mammary gland.*—Normal. *Supramammary lymph gland.*—Normal. *Ovary.*— Left.—Fibrous filaments, cysts of Graafian follicle type. Right. — Cysts of Graafian follicle type. *Fallopian tubes.*— Left and Right.—Hydrosalpinx which is local, confined to one portion of the tube. *Uterus.*—Endometritis chronica catarrhalis cystica. *Vagina.*—Normal. *Vulva.*—Lymphocytic hyperplasia.	23.10.25: Cyst expressed from the right ovary. Corpus luteum expressed from the left. 26.10 25: Animal shows oestrum and is served. 12.2.26: Animal has not been in oestrum, and gross examination reveals cystic ovary again developed and cystic degeneration of the uterine wall. Case considered hopeless and recommended for destruction.	Cystic degeneration of the cervical mucosa. endometritis catarrhalis chronica. salpingitis, and cystic oophoritis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis.................................. —
Hypophysis, 2·6 × 1·5 × 1·7 cm........ Weight 4·6 gms.
Adrenals—Right, 7·2 × 4·3 × 1·5 cm... ,, 25·5 ,,
Left, 4·7 × 5 × 2 cm......... ,, 22·7 ,,

Thyroid—Right, 5·8 × 5·5 × 1 cm.
Left, 5·2 × 6 × ·8 cm.
Length, including isthmus, 20 cm., weight 20·6 gms.

CASE 33. (See Appendix II, Fig. 32.)

AYRSHIRE COW 1808, 7 YEARS OLD.

Date of first examination, 23.10.25. *Date of slaughter*, 5.11.25

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 919 lb. Cow appears normal. The udder is non-lactating. No. of calves—3. Last parturition—July, 1924, since when periods of oestrum have been irregular. Contagious abortion test—positive. Tuberculin test—positive.	Vulva shows old lesions of granular vaginitis. The vagina contains a quantity of thick mucus, with greyish - yellow flocculi. The first cervical fold is ectropic and ragged. The mucosa is dark red in colour. The circumference of the cervix is greatly enlarged. Through the rectum it appears firm. The uterus is mostly abdominal; both it and the uterine horns are enlarged. Its walls are thick and there is a nodular feel. It is fluctuating and contains a quantity of liquid. The fallopian tubes are markedly enlarged to the size of a lead pencil. They are convoluted and firm throughout their entire extent. The fimbriated extremity of both tubes is partly adherent to the ovary. Right ovary size of a small hen egg, oval in shape; its surface is irregular and fluctuating. Left ovary, size of a large walnut, but somewhat elongated: it is normal in consistency. The broad ligaments appear somewhat elongated. *Diagnosis:* salpingitis, cystic ovary, cervicitis, tubercular pyometritis.	Vulva shows some yellow-grey transparent nodules. The vagina contains some thick mucus mixed with greyish - yellow flocculi. Cervix, 6·5 cm long; mucosa is red in colour; shows an ectropic first fold. The second fold is elongated and protrudes through the posterior opening of the os. The cervical wall is thickened. The uterus is enlarged — body, 40 c.m.; left horn, 12 × 11 cm.; right horn, 36 cm. long. It shows on its serous surface numerous fibrous adhesions especially towards the apices of the horns. The wall is firm and granular; the mucosa is studded with yellowish tubercles from 1 mm. to 1 cm. in diameter; these are raised above the surface of the mucosa, which is swollen and reddish throughout. There is no macroscopic evidence of normal cotyledons. The uterus contains a yellowish exudate which distends the uterus to a fair extent. It is cream-like in consistency. The tubercles are confined to the surface of the walls, but some extend into the muscular portion. The fallopian tubes are 28-30 cm. long, firm, cord-like, and convoluted; the greatest thickness is at the centre, where they are 1·2 cm. in diameter. The serous coat shows some fibrous filaments. On section, they are filled with yellow caseous material. The fimbriated extremity is attached to the ovary. P.-m. showed tubercular lesions in the thorax and abdomen.	Right ovary, 4 × 2·5 × 1·5 cm. The surface shows fibrous filaments. On palpation, the ovary is cystic. On section, it contains a large cyst 2·5 cm. in diameter and a small cyst ·75 cm. Both cysts are filled with a clear amber fluid. On the border of the smaller cyst traces of lutein tissue can be seen. There are also present two cavities, ·6 and ·8 cm. in diameter, which contain what appears to be clotted blood. The remainder of the ovary is whitish-grey in colour. No trace of recent corpora lutea can be seen. Left, 4·5 × 3 × 2·5 cm. The surface shows fibrous thickenings irregularly distributed over it. It shows no trace of recent corpora lutea on the surface. It appears normal in consistency. On section, there are numerous cyst - like structures arranged close to the surface. These vary up to 1 cm. in diameter.	*Cervix,*— Cervicitis. chronica catarrhalis with some sclerosis. *Vagina.*—Normal. *Uterus.*—Metritis tuberculosa. *Fallopian tubes.*—Salpingitis tuberculosa. *Ovaries*—. Right.—Cyst, Graafian follicle type. Left.—Normal follicles. *Adrenal glands.*—Normal. *Epiphysis.*—Few areas of calcification. *Hypophysis.*—Normal. *Thyroid.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal.	No treatment tried. Prognosis hopeless.	Cervicitis, tubercular metritis, salpingitis, cystic oophoritis.

SIZE AND WEIGHTS OF ENDOCRINES.

Adrenals—Right, 7 × 4 × 1·25 cm...... Weight 16·25 gms.
Left, 5·5 × 4 × 1·5 cm....... ,, 15·2 ,,

Thyroid—Right half, 5·5 × 6 cm.
Left half, 4·5 × 6 cm.
Length, including isthmus, 22 cm., weight 24 gms.

CASE 34. (See Appendix II, Fig. 33.)

FRIESLAND COW 1870, 9 YEARS OLD.

Date of first examination, 9.2.26. *Date of slaughter*, 29.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1.100 lb. There is slight relaxation of the pelvic ligaments. The udder has lactated, but is now dry. Mammary tissue is soft and pliable. The cow has suffered from contagious granular vaginitis. No. of calves, 2. Date of last parturition, October, 1923. While under observation at this Institute for 4½ months, the cow showed oestrum six times, but the intervals between oestral periods were somewhat irregular. There was no evidence of sexual abnormality between oestral periods. Contagious abortion test, negative Tuberculin test, positive.	The vulva shows a pale mucosa with a few yellowish semi-transparent nodules. The vagina is normal. The cervix shows a pale mucosa; it is transversed by a vertical band, which divides its posterior extremity in two parts. Both openings admit the finger to a depth of about 4 cm.; it is impossible to say whether the band divides the cervix throughout its entire length. On rectal examination the cervix appears normal in length and circumference. Uterus—body appears normal in size and tone. The left horn is smaller than the right, and appears thin-walled; it, however, is of good tone. The right horn is somewhat larger than the left and appears of normal tone. The right fallopian tube is enlarged. Its fimbriated extremity is attached to the ovary by its free border and forms a large soft cyst. The left fallopian tube is also enlarged and its fimbriated extremity also forms a soft cyst. The right ovary is the size of a hen's egg and markedly fluctuating. The left ovary is enormously enlarged about the size of a duck's egg; it is fluctuating and thin-walled throughout. There are some loose adhesions between the broad ligaments and the uterus. *Diagnosis*: Cervix duplex, cystic ovaries, hydrosalpinx, pavilionitis.	The vulva shows some yellowish nodules, mucosa pale. Vagina mucosa pale. Cervix—length, 9 × 7 cm. Width of band—posteriorly, 1 cm.; anteriorly, ·5 cm. Mucosa pale. The cervix is divided into two portions by a longitudinal vertical band which passes throughout its entire length. Both ostia admit the small finger for a distance of 4 cm.; they gradually decrease in calibre towards their anterior extremity; both open freely into the pars indivisa of the uterus. The uterus—length, 10 × 6 cm.; width of wall, 1·2 cm.; left horn, 18 × 2·5 cm.; width of wall, ·7 cm.; right horn, 20 × 3·5 cm.; width of wall, 1 cm. The organ is fairly firm. The serous surface is yellowish-pink in colour and shows some loose fibrous adhesions to the rectum and broad ligaments. On section, the mucosa of the pars indivisa and both horns, are greyish-pink: mucosa smooth and moist. The cotyledons are small in size and of a greyish-yellow colour. The fallopian tubes are dilated throughout their entire length. The left is up to 1·5 cm., the right up to 2·5 cm. in diameter. They are thin-walled and contain a clear fluid. The fimbriated extremity of each tube is attached by its free border to the corresponding ovary. Each forms a large soft cyst; on the left side it reaches 6 × 4 cm., on the right side 7 × 5 cm.; both are thin-walled and contain clear fluid. The broad ligaments show some fibrous attachments. P.-m. showed tubercular lesions present in the bronchial lymphatic glands.	The right ovary, 4·5 × 4 × 4 cm. Its surface is smooth except where it is attached to the free border of the fimbriated extremity of the fallopian tube. The ovary is fluctuating on palpation. Its capsule is thin-walled. On section, shows a central corpus luteum 1·8 cm. in diameter. There are several atrophied corpora lutea present. There are three cysts; these contain a clear fluid. The ovarian tissue appears greyish-white in colour and firm in consistency. The left ovary, 8·5 × 5 × 5 cm.: surface shows numerous fibrous filaments. It is fluctuating throughout and many portions of its capsule are semi-transparent. On section, it contains a single large cyst with a very thin capsule. The ovarian tissue has undergone complete atrophy. The contents of the cyst are thin brownish liquid which is non-viscid.	*Thyroid gland.*—Normal. *Pancreas.*—Normal. *Thymus.*—Normal. *Adrenal glands.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vagina.*—Lymphocytic accumulations. *Cervix.*—Mucosa normal. *Uterus.*—Perimetritis, chronic changes in mucosa, oedema, thickening of walls of the vessels, with some hyaline degeneration, glands enlarged. *Fallopian Tubes.*—Hydrosalpinx. *Ovaries.*— Right.—Cysts lined with epithelum. corpus luteum with loose fibrous tissue mesh towards its centre. Left.—Large cyst with epithelial lining.	Case hopeless. No treatment tried.	Metritis chronica catarrhalis, hydrosalpinx, pavilionitis. cystic ovary.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·2 × ·6 × ·6 cm.	Weight 0·3 gm.	Thyroid—Right half, 7·4 × 4·5 × 1·2 cm.
Hypophysis, 2·3 × 1·8 × 1·3 cm.	,, 3·0 gms.	Left half, 6 × 4·2 × 1·1 cm.
Adrenals—Right, 8 × 3·1 × 1·3 cm.	,, 15·0 ,,	Length, including isthmus, 21 cm., weight 34·5 gms.
Left, 4·5 × 3·8 × 1·1 cm.	,, 14·0 ,,	

CASE 35. (See Appendix II, Fig. 34.)

FRIESLAND COW 1871, 4 YEARS OLD.

Date of first examination, 9.2.26. *Date of slaughter*, 1.7.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,100 lb. The head is slightly coarse, otherwise the appearance is normal. The udder has not lactated. The animal has never been pregnant although frequently bred. The cow has had contagious granular vaginitis, but has never been treated. Contagious abortion test, negative. Tuberculin test, positive.	The vulva is small; it does not admit the hand; the mucosa shows numerous small elevated nodules and is generally of a reddish colour. The vagina cannot be examined internally. From the rectum it appears normal. Cervix is normal in size and consistency. The uterus is slightly smaller than normal, and is of good tone. Both uterine horns are also small, but normal in tone. The dorsal and lateral aspect of the uterus and horns have got some loose fibrous filaments attached. These extend between the uterus, broad ligament, the rectum, and the ovary. The right fallopian tube is enlarged, elongated and tortuous; it contains fluid. The fimbriated extremity is very much enlarged and its free border attached to the ovary; it forms a soft thin-walled cyst. The left fallopian tube is also enlarged and tortuous. Its fimbriated extremity is attached to the ovary and forms a small soft cyst. The right ovary is firm, the size of a small hen egg. The left ovary is firm, somewhat smaller than the right. Both ovaries are difficult to palpate; they seem to be involved with fibrous adhesions. *Diagnosis*: hydrosalpinx pavilionitis. While under observation a cyst developed in the left ovary.	The vulva shows some nodules on the mucosa. The vagina is normal. Cervix, 7 × 4·5 cm.; width of wall, 2 cm. It is anatomically normal, resembling the cervix of a virgin heifer. Uterus, 8 × 5 cm.; right horn, 18 cm.; left, 16 cm.; width of wall of uterus, ·6 cm.; width of wall of horns varies from ·6–·4 cm. The serosa of the uterus is mostly covered with loose fibrous adhesions, which pass between it and the rectum, the broad ligaments, and the bladder. Where adhesions are absent the serosa is of a yellowish-pink colour. On section, the mucosa is greyish-pink with a slightly yellowish tinge; it is smooth and moist. There is no evidence of cotyledons. There are several small depressions varying in size from 1–2 mm. and ·5 mm. in depth. Both fallopian tubes are enlarged and convoluted; they contain a clear fluid. The fimbriated extremity of both tubes is cystic and attached to the corresponding ovary. On the right side it is 9 × 7 × 5 cm. and contains a darkish-yellow fluid with a slightly reddish tinge. On the left side it is 2·7 × 2 cm. and contains a clear fluid with a slightly reddish tinge. Both broad ligaments show loose fibrous adhesions. Post-mortem showed generalized tuberculosis.	The left ovary, 4·5 × 3·4 × 2·3 cm. It is encapsulated in connective tissue. Its anterior pole is slightly fluctuating; its posterior pole is firm. It is adherent to the left horn of the uterus through a thick band of connective tissue; bands also pass between it and the fallopian tube. On section, it shows a central corpus luteum 2·5 × 2 cm. of an orange-yellow colour. There are six small cyst-like bodies varying in size from ·3 cm. to 1·8 cm. in diameter. These contain a clear fluid. The remaining portion of the ovary is greyish-white and firm in consistency. The right ovary, 3 × 3 × 2·5 cm. It is closely adherent and the fallopian tube and its fimbriated extremity. It is encapsuled in connective tissue. On palpation, it appears flabby but not fluctuating. This is due to encapsulation with connective tissue. On section, there is a thick connective tissue capsule ·4 cm. in width. There are three atrophied corpora lutea evident. There are two small cyst-like areas, ·3 cm. in diameter. The remaining portion of the ovary is greyish-white and firm.	*Epiphysis.*—Corpora amylacea, arteriosclerosis. *Hypophysis.*—Normal. *Adrenals.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Metritis chronica catarrhalis. *Fallopian tubes.*—Hydrosalpinx. *Ovaries.*— Right.—Small cyst. 240 × 80 *m*, with low cubical epithelium and a connective tissue capsule. Left.—Several small cysts lined with epithelium—size 80 × 120 *m*; epithelium lies on a membrane of connective tissue. Some normal follicles. Lutein tissue in corpus appears normal.	Case hopeless. No treatment tried.	Metritis chronica catarrhalis, hydrosalpinx, cystic ooporitis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·5 × ·5 × ·5 cm..........	Weight 0·3 gm.
Hypophysis, 2 × 1·5 × 1 cm...........	,, 2·0 gms.
Adrenals—Right, 5 × 3·6 × 1·3 cm.....	,, 13·0 ,,
Left, 4·2 × 4 × 1·4 cm.......	,, 11·0 ,,

Thyroid—Right half, 6 × 3·4 × 1·2 cm.
Left half, 5·4 × 3 × ·9 cm.
Length, including isthmus, 22., weight 27 gms.

CASE 36. (See Appendix II, Fig. 35.)

FRIESLAND COW 2071, 5 YEARS OLD.

Date of first examination, 15.4.26. *Date of slaughter*, 15.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition poor. Weight 800 lb. The cow is normal in appearance. The udder has lactated, but is now dry. It is normal on palpation. No. of calves, 2. Date of last parturition. 1/3/24 (abortion). Since last parturition the cow has not shown oestrum. There is no history of contagious vaginitis. The animal was not tested for abortion until 15/4/26, when the test was negative. This was more than two years after the cow aborted. Tuberculin test. negative.	The buttocks are stained with thick yellow pus. The mucosa of the vulva is normal. The vagina contains a quantity if tenacious pus mixed with mucus. The posterior extremity of the cervix is ectropic and is reddish in colour. A small quantity of purulent material issues from the os. On rectal examination, the cervix appears somewhat increased in diameter, although it is not abnormally firm. The uterus is abdominal; it is thick-walled and fluctuating. The uterine horns are also thick-walled and fluctuating, and of equal size. The uterine arteries are not increased in size. Both fallopian tubes are normal in size and consistency. Left ovary normal in size and consistency; it contains a corpus luteum, which is prominent, towards its anterior pole. Right ovary normal in size, firm in consistency. The broad ligaments are slightly elongated. *Diagnosis*: pyometra, with retained corpus luteum. There was little change in the condition during the time the cow was kept under observation.	The vulvar mucosa is normal. The vagina contains a quantity of thick, slightly greenish-yellow foetid pus. The first cervical fold is slightly protruding. The mucosa of the cervix is pale pink in colour. On section—length, 8 cm.; width posteriorly, 5 cm.; anteriorly, 4·2 cm.; width of wall, 2·5 cm. posteriorly, 1·9 cm. anteriorly. Attached to the cervix between its folds is a tough semi-transparent tenacious mucus of the consistency of the cervical plug of pregnancy. The consistency of the cervix appears normal. Uterus, 12 × 8 cm.; horns, 19 × 6 cm. The serous surface is pale, yellowish-pink in colour and smooth. The organ is fluctuating and distended; both horns are equal in size, with somewhat rounded apices. On section, it contains two litres of thick, greenish-yellow foetid pus. The wall is firm and varies in thickness from 1·4 × 1·8 cm. The mucosa of the horns is entirely changed. It is of a mottled greyish-red colour. There are a few small irregular reddish areas towards the apices of the horns. The lining has a granular feel, the granules vary up to 3 mm. in diameter and are prominent. There are a few whitish rounded nodules, from 1 to 4 mm. in diameter, attached to the lining of the horns through a small neck. On section, these are white and firm. Immediately in front of the cervix, the uterine body shows a smooth, pale pink mucosa without evidence of folds or cotyledons. The fallopian tubes are normal in size and consistency.	Right, 3·8 × 2 × 1·7 cm. Shows on its surface some Graafian follicles in various stages of development. On section, several apparently normal Graafian follicles are present: some atrophied corpora lutea are also seen. The ovarian tissue is greyish-white in colour and apparently normal. Left, 4 × 2·8 × 2 cm. Shows a large prominent corpus luteum and Graafian follicles in various stages of development, the largest of which is 1·4 cm. in diameter. On section, there is a large corpus luteum, 2·4 × 1·7 cm., of a pale orange colour. There are also several atrophied corpora lutea of former interovulation periods present. Several apparently normal Graafian follicles are seen; the largest is 1·2 cm. The ovarian tissue is whitish-grey and normal in consistency.	*Hypophysis.*—Some vesicles filled with colloid in the centre of the pars distalis. *Epiphysis.*—Some small corpora amylacea. *Adrenals.*— Right.—Normal. Left.—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*— Mastitis chronica catarrhalis, with some fibrosis of the insterstitial connective tissue. *Cervix.*—Slight fibrosis. *Uterus.*—Pyometra. with disappearence of epithelium, and fibrosis of the mucosa with reduction of the mucous glands, also commencing cystic formation in some of the glands. *Fallopian tubes.*— Salpingitis chronica with fibrosis of the villi and the wall. *Ovary.*— Left.—Some normal follicles, small cystic degeneration. Corpus luteum of last interovulation period, normal. Right.—Several atretic follicles and some normal follicles.	Case considered hopeless. No treatment tried.	Pyometra.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·1 × ·5 × ·3 cm...........	Weight 0·2 gm.	Thyroid—Right half, 4·5 × 6 × ·7 cm.	
Hypophysis, 2·2 × 1·8 × ·9 cm.........	,, 2·5 gms.	Left half, 5·2 × 3·5 × ·9 cm.	
Adrenals—Right, 6·2 × 4·3 × 1·4 cm...	,, 17·5 ,,	Length, including isthmus, 24·2 cm., weight 25 gms.	
Left, 4·3 × 4·2 × 1·6 cm....	,, 15·5 ,,		

CASE 37. (See Appendix II, Fig. 36.)

FRIESLAND COW 2073, 8 YEARS OLD

Date of first examination, 20.4.26. *Date of slaughter*, 17.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 950 lb. There is slight relaxation of the pelvic ligaments on either side of the root of the tail and the sacrum. The head is somewhat coarse in appearance. The udder is non-lactating. The cow has calved several times. Date of last parturition —2·2·1924, since then the periods of oestrum have become irregular. The cow has been bred several times without conceiving. The animal has suffered from contagious granular vaginitis. Contagious abortion test—negative Tuberculin test—positive.	The vulva shows some nodules of granular vaginitis, in a dormant stage. The mucosa appears pale in colour. The vagina is normal. The cervix is normal in colour. Its posterior extremity is not abnormally ragged for a cow, which has had several parturitions. It is of normal size and consistency. The uterine body is of good tone and normal in size. The uterine horns are abdominal, equal in size and of good tone. The fallopian tubes are enlarged, especially towards the ovarian extremity, where they appear to be thin-walled and cystic. The fimbriated extremity of both tubes is attached to the ovary. It is thin-walled and cystic. Right ovary is the size of a small orange. It is rounded and fluctuating. The left ovary is the size of a walnut, oval in outline, and feels fluctuating in parts. Broad ligaments are normal. *Diagnosis*: cystic ovary. hydrosalpinx.	The vulva shows some dormant nodules of granular vaginitis; its mucosa is pale bluish in colour. The vagina is normal. Cervix, 5·5×4·5 cm.: width of wall, 2 cm. The first cervical fold is somewhat protruding and slightly ragged. The mucosa is pale pinkish in colour; the consistency is normal. Uterine body, 10·5× 4·5 cm., width of wall, 1·6 cm.; uterine horns—left, 19·5 cm.; right, 17·5 c.m.: width, 3 cm. width of wall, 1·25 c.m. The serosa is yellowish-red in colour and smooth, except towards the apices of the horns and two small areas on the lateral face of either horn, where it is adherent to the ovary and meso-salpinx through fibrous tissue connection. On section, the uterine wall is somewhat thickened; its mucosa is dull greyish-red in colour, markedly swollen and oedematous. There is no evidence of normal cotyledons. The summit of the uterine folds shows small depressions oval in shape, varying in size up to 4 mm. in length and 1 mm. deep. Both fallopian tubes are very much increased in, size especially towards the fimbriated extremity, where they reach a width of 2 cm. They are tortuous, thin-walled, and cystic; they contain a clear fluid. The fimbriated extremity of each tube is adherent to the corresponding ovary all round its border. Each forms a thin-walled, soft cyst about 4 cm. in diameter. The broad ligaments are normal. Post-mortem showed the presence of tubercular lesions in the bronchial and mediastinal lymphatics and the lung tissue.	Right ovary, 4·5×4·5×4·5 cm. It is adherent to the free border of the fimbriated extremity of its tube, and by its posterior pole to the lateral face of the right horn of the uterus, through a band of connective tissue. The ovary is rounded and fluctuating throughout; its surface presents a small cyst-like structure 1 c.m. in diameter on its medial aspect. On section, this contains a clear fluid. On section, the ovary contains a large single cyst, with transparent, slightly yellowish fluid contents. Its wall is smooth and contains a thin band of lutein tissue throughout. The ovarian tissue has undergone almost complete atrophy, the cyst capsule being a few millimetres in thickness. Left ovary, 3·5×2·5×2·4 cm., is partly encapsuled by connective tissue, through which it is adherent to the meso-salpinx, the apex of the horn of the uterus, and the lateral face of the left horn of the uterus. It shows several fluctuating areas over which the capsule is somewhat transparent. On section, there are several cyst-like vesicles, varying in size, some peripheral, some central, and six atrophied corpora lutea. The ovarian tissue is greyish-white in colour and firm.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland*—increase in connective tissue. *Cervix.*—Sclerosis. *Uterus.*—Endometritis chronica catarrhalis, with commencing cystic formation. *Fallopian tubes.*—Hydrosalpinx. *Ovary.*— Right.—Corpus luteum cyst. Left.—Normal follicles present.	Case considered hopeless. No treatment tried.	Metritis chronica catarrhalis, hydrosalpinx, cystic ocphoritis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·2 × ·4 × ·4 cm.	Weight 0·15 gm.	Thyroid—Right half, 4·5 × 4·8 × 1 cm.
Hypophysis, 2·5 × 1·6 × 1·3 cm.	„ 3·0 gms.	Left half, 4·4 × 3·8 × 1 cm.
Adrenals—Right, 4·2 × 5·5 × 1·2 cm.	„ 14·5 „	Length, including isthmus, 29·2 cm., weight 24·5 gms.
Left, 4·7 × 4 × 1·1 cm.	„ 13·5 „	

CASE 38. (See Appendix II, Fig. 37.)

FRIESLAND COW 2074, 10 YEARS OLD.

Date of first examination, 20.4.26. *Date of slaughter*, 22.6.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 950 lb. The pelvic ligaments are slightly relaxed. The root of the tail is somewhat elevated. and the rump on either side of the sacrum is slightly hollow. The udder is not lactating. Its tissue is normal in texture. Number of calves—several. Last parturition—23.7.1924. Was in very poor condition at last parturition. Since last parturition the cow had frequently been bred without success. During the period of two months during which the animal has been under observation she has not shown oestrum. Contagious abortion test—negative. Tuberculin test—negative.	The vulva shows some dormant lesions of granular vaginitis, mucosa pale. The vagina contains some thick tenacious transparent mucus. The pars vagina of the cervix is ragged and slightly reddish in colour. The first cervical fold is ectropic and dark red in colour. The os uterus admits the small finger to a depth of 1¼ inches. Per rectum, the cervix appears to be enlarged posteriorly. Its anterior extremity is normal in circumference. The uterus is mostly abdominal, normal in size, and firm in consistency. The uterine horns are normal in size and firm in consistency. The fallopian tubes are irregularly enlarged along their entire length. Some of the enlargements are rounded. others elongated. The fimbriated extremity of each tube is attached to the corresponding ovary. The extremity is enlarged, fluctuating and soft. The right ovary is the size of a walnut and firm in consistency. The left ovary is rounded, normal in size and firm in consistency. The broad ligaments are normal. *Diagnosis*: cervicitis and salpingitis.	The vulva shows some dormant lesions of granular vaginitis. The vagina is normal. Cervix, 9·5 × 6 × 4 cm. The posterior extremity of the cervix is ragged. The first cervical fold is protruding and slightly reddish in colour. The os uterus is patent. Uterus—length, 10 × 5 cm.; length of horns, 20 cm.; width, 3·5 cm. On section, thickness varies from 1·3–1 cm. The serosa of the uterus is smooth and yellowish-pink in colour. Consistency is firm. The mucosa is greyish-pink in colour and moist. Cotyledons are prominent, whitish-grey to pinkish-white in colour. Both fallopian tubes are irregularly enlarged towards their cornual extremity. They are cystic, thin-walled, and contain a clear fluid. The middle portion of both tubes is normal in size and consistency. Towards the abdominal extremity, they are fairly thick-walled, fluctuating, yellowish in colour, and contain yellow, purulent material. The fimbriated extremity of both tubes shows the free border of the funnel adherent, forming a closed cavity, which is filled with yellow purulent liquid material. The broad ligaments are normal.	Right ovary, 3·4 × 2 × 2 cm. It is firm in consistency. Traces of lutein tissue can be seen on its surface. On section, shows seven atrophied corpora lutea of previous interovulation periods; they are all practically the same size. The ovarian tissue is greyish-white and firm. There is no macroscopic evidence of developing Graafian follicles Left ovary, 4·5 × 3·5 × 3 cm., shows several small areas on its surface, which contain liquid and over which the capsule is semi-transparent. It shows traces of lutein tissue on its surface, on section, shows a large central corpus luteum 2·5 cm. in diameter. It is pale yellowish in colour; Its centre contains a cyst 1·8 cm. in diameter. The capsule of the cyst is whitish in colour—that is, the corpus luteum merges from pale yellow to white. The cyst contains an opaque slightly viscid fluid. The remaining ovarian tissue is greyish-white in colour and firm. There is no macroscopic evidence of developing Graafian follicles.	*Epiphysis.*— Corpora amylacea. Size, 5 × 7 m. to 124 × 88 m. *Hypophysis.*— Hyperaemia of pars distalis. Two vesicles, (1) 368 × 340 *m.* (2) 136 × 126 *m.*; both are lined with low cubical epithelium, and filled with colloid. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland*—Normal. *Adrenals.*—Normal. *Vagina.*—Normal. *Cervix.*—Fibrosis. *Uterus.*—Metritis chronica catarrhalis with commencing cystic formation. Some hyaline degeneration of vessel walls. *Fallopian tubes*—Hydrosalpinx. *Ovaries.*— Right.— Sclerosis, Primary and secondary follicles. Left.—Primary and secondary follicles, corpus luteum cyst. Wall three layers (*a*) outside of fibrous tissue (*b*) middle of lutein tissue (*c*) inner layer of connective and fibroblastic tissue.	Case considered hopeless. No treatment tried.	Cervicitis, salpingitis, microscopic evidence of metritis chronica catarrhalis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·5 × ·5 × ·5 cm.	Weight 0·4 gm.
Hypophysis, 2·4 × 1 6 × 1·2 cm.	,, 3·0 gms.
Adrenals—Right, 6·5 × 4·5 × 1·4 cm. ...	,, 18·5 ,,
Left, 5·8 × 3 × 1·3 cm.	,, 13·5 ,,

Thyroid—Right half, 4·8 × 5·6 × ·6 cm.
Left half, 5 × 3·5 × ·8 cm.
Length, including isthmus, 21 cm., weight 13·5 gms.

CASE 39. (See Appendix II, Fig. 38.)

FRIESLAND COW 2110, 6 YEARS OLD.

Date of first examination, 3.8.26. *Date of slaughter*, 23.9.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 980 lb. Normal in appearance. The udder has lactated, but is now dry. Udder tissue normal. No. of calves—2, both abortions. Date of last abortion—January, 1924. Has been served several times without results. Oestral periods normal: while under observation at this Institute the cow had four oestral periods at normal itenrvals. Contagious abortion test—positive. Tuberculin test—negative.	There are numerous enlarged lymph follicles in the vulva. Vagina is stenosed about 8 cm. caudal to the cervix, so that it admits only two fingers. The cervix is examined with difficulty; it does not appear changed. There is a small quantity of tenacious mucus in the anterior portion of the vagina. From the rectum the cervix is normal in size and consistency. Uterus is mostly abdominal. The left horn is somewhat larger than the right; it appears normal in consistency and of good tone. There are some fibrous adhesions between its dorsal surface and the rectum. Fallopian tubes: right, is dilated throughout its entire length. Its fimbriated extremity is adherent to the ovary and forms a soft cyst. The left is dilated throughout its entire length. Its fimbriated extremity is adherent to the ovary, and at its junction to the ampulla, there is a firm cystic enlargement about the size of a marble. Right ovary size of a pigeon's egg, encapsulated in fibrous tissue, difficult to palpate. It appears firm in consistency. Left ovary size of a pigeon's egg, also encapsuled in fibrous tissue, difficult to palpate, appears firm in consistency. Broad ligaments normal. *Diagnosis*: metritis chronica catarrhalis, hydrosalpinx, pavilionitis.	Vulva shows some swollen lymph follicles. The vagina is stenosed about 8 cm. behind the cervix. The stenosis is due to a cicatrix in the wall, probably a result of parturition injury. Cervix, 7 × 5 cm.; width of wall, 2 cm.; mucosa pale-pink in colour. The cervical folds are very well marked, the posterior two being perhaps slightly hypertrophied. Uterus—body, 10 × 4 cm; width of wall, 1 cm.; left horn, 20 × 2·5 cm.; width of wall, 1·8 cm.; right horn, 19 × 2·5 cm., width of wall, 1·8 c.m. The serosa of the uterus is pale pinkish-yellow in colour. There are some loose fibrous adhesions between it, the rectum, and the broad ligaments. The mucosa is pale pinkish-grey with a slightly yellowish tinge. It is moist and somewhat swollen. There are no normal cotyledons. Their position is represented by a yellowish-grey area with a small central depression. Both fallopian tubes are distended throughout their entire length, up to 7 mm. in diameter. The ampulla of both tubes reaches up to 1·5 cm. The fimbriated extremity of both tubes is attached to the corresponding ovary. It forms a cyst with a thin, more or less transparent capsule. That on the right side is 2 cm. in diameter, on the left 4 cm. in diameter. Both tubes contain a slightly opaque liquid with some minute flocculi.	Right, 2·5 × 2·5 × 2 cm. It is entirely encapsuled by the fimbriated extremity of the fallopian tube and fibrous tissue, through which it is adherent to the meso-salpinx. Its surface is not visible. On section, there are traces of five atrophied corpora lutea. The corpus luteum of the second last interovulation period is present. The remainder of the ovarian tissue is greyish-pink and firm. Left, 3·5 × 2·7 × 2·7 cm. It is entirely encapsuled by fibrous tissue and the fimbriated extremity of the fallopian tube. There are fibrous adhesions between the ovary and the mesosalpinx. Its surface is not visible. On section, there is a large embedded corpus luteum with a large central cyst. The capsule of ovarian tissue is 3 mm. in thickness. It is firm greyish-pink to greyish-yellow in colour.	*Epiphysis.*—Normal. *Hypophysis.*—Vesicle, filled with colloid, lined with cubical epithelium, in pars distalis. *Adrenals.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland.*—Normal. *Vagina.*—Normal. *Mammary gland.*—Slight catarrhal mammitis. *Cervix.*—Normal. *Uterus.*—Chronic changes, mucosa shows fibrosis and some thickening of the vessel walls. *Ovaries.*—Atretic follicles, sclerosis, corpus luteum cyst, corpus albicans with central cavity containing fluid, oedema some normal follicles.	Case hopeless. No treatment tried	Metritis chronica catarrhalis, hydrosalpinx, pavilionitis, cystic corpus luteum.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·5 × ·6 × ·6 cm. .... Weight 0·2 gm.
Hypophysis, 2·5 × 1·8 × 1·2 cm. .... „ 3·0 gms.
Adrenals—Right, 4·8 × 3·9 × 1·2 cm. ... „ 11·5 „
Left, 6·1 × 3·5 × 1·3 cm. .... „ 12·7 „

Thyroid—Right half, 5·5 × 5·2 × 1·2 cm.
Left half, 5 × 4·3 × 1·2 cm.
Length, including isthmus, 19 cm., weight 25·5 gms.

CASE 40. (See Appendix II, Fig. 39.)

FRIESLAND COW 2111, 8 YEARS OLD.

Date of first examination, 3.8.26. *Date of slaughter*, 16.9.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,000 lb. Normal in appearance. The udder is in a later stage of lactation. Tissue normal. No. of calves—3, including one abortion in 1923. Last parturition—July, 1925. Cow has been served several times without result. Contagious abortion test—negative. Tuberculin test negative. She suffered from infectious vaginitis in 1925. While under observation the oestral periods were irregular with prolonged interovulation period.	Vulva shows numerous dormant enlarged lymph-follicles. Vagina stenosed about 3 inches caudal to the cervix so that it admits only 2 fingers with difficulty. The cavity in front of the stenosis is filled with thick tenacious mucus containing some white flocculi. Cervix can just be felt, its posterior fold is ectropic. From the rectum the cervix is normal in size and firm in consistency. The uterus and uterine are soft and flaccid, lacking in tone. The left horn is larger than the right. The right fallopian tube is enlarged, firm, and markedly tortuous. The fimbriated extremity is adherent. Left fallopian tube is normal. Right ovary size of a pigeon's egg, firm in consistency. It is adherent to the meso-salpinx and the fallopian tube. Left ovary somewhat larger than a walnut, firm in consistency; shows the presence of a well-developed Graafian follicle. Broad ligaments are normal. *Diagnosis*: Vaginal stenosis metritis chronica catarrhalis, hydrosalpinx.	Vulva shows some enlarged lymph follicles. Mucosa pale. Vagina shows a cicatricial stenosis 3 inches behind the cervix; it contains some tenacious mucus. Cervix, 8 × 6 cm.; width of wall, 3 cm. First cervical fold ectropic; mucosa pale. Uterus—body, 12 × 6 cm.; width of wall, 1 cm.; left horn, 25 × 4 cm.: width of wall, 1–·8 cm.; right horn, 23 × 2·5 cm.; width of wall, 1–·8 cm. The wall is soft and flabby. The serosa is greyish-pink in colour, smooth, and, on section, the mucosa is very moist, swollen, and greyish-pink in colour. There is no trace of cotyledons in the right horn. In the left horn there are some cotyledonary remains which are yellowish-white in colour; they are irregular in size and do not project above the level of the surrounding mucosa. The left fallopian tube is normal. The right fallopian tube is markedly enlarged throughout its entire length. It is tortuous, and, on section, contains an opaque greyish fluid. The tube and the mesosalpinx are connected by fibrous adhesions to the ovary and the horn of the uterus. The fimbriated extremity is adherent, but not cystic. Broad ligaments normal. Carcase showed tuberculosis in mesenteric lymphatics.	Left ovary, 3·5 × 2·8 × 2·4 cm. It is oval in shape, shows traces of several atrophied corpora lutea and numerous Graafian follicles. There is a prominent corpus luteum present. On section, the ovarian tissue is greyish-white There is a small atrophied corpus luteum, and the corpus luteum of the last interovulation period, 1·5 × 1·2 cm., present. There is also a normal Graafian follicle ·6 cm. in diameter. Right ovary, 3·6 × 2·5 × 2·5 cm. It is surrounded by dense fibrous tissue which occludes the surface of the ovary. On palpation, it is firm. It is adherent to the fimbriated extremity of the fallopian tube and the mesosalpinx, through fibrous adhesions. On section, traces of lutein tissue are evident. There is a small central cyst-like structure ·4 cm. in diameter. The ovarian tissue is greyish-white and firm. There is no evidence of a superficial ripening-follicle.	*Epiphysis*.—Normal. *Hypophysis*.— Small vesicle in pars intermedia. *Thyroid*.—Normal. *Thymus*.—Normal. *Adrenals*.—Normal. *Supramammary lymph gland*.—Normal. *Mammary gland*.—Normal. *Pancreas*.—Normal. *Vagina*.—Normal. *Cervix*.—Slight sclerosis. *Uterus*.—Endometritis chronica catarrhalis. *Fallopian tubes*.— Right.—Hydrosalpinx, perisalpingitis. Left.— Catarrhal salpingitis in early stages. *Ovary*.— Left.—Normal. Right.—Fibrous adhesions on the surface of the right ovary.	Case considered hopeless. No treatment tried.	Metritis chronica catarrhalis. hydrosalpinx.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·2 × 1·5 × 1·5 cm........ Weight 3·0 gms.
Epiphysis, 1·2 × ·5 × ·5 cm............ „ 0·5 gm.
Thyroid—Right. half, 5 × 4 × ·9 cm.
Left, 5 × 4·7 × 1 cm.
Length, including isthmus, 19 cm......... „ 22·5 gms.
Adrenals—Right, 4·5 × 4·5 × 1·4 cm. Weight 1315 gms.
Left, 5·5 × 4·9 × 1·2 cm., „ 16·0 „

CASE 41. (See Appendix II, Fig. 40.)

FRIESLAND COW 2112, 6 YEARS OLD.

Date of first examination, 8.7.26. *Date of slaughter*, 7.9.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,000 lb. Normal in appearance. Has lactated, but is now dry. There is a fracture of the second coccygeal vertebra, which causes a depression at the root of the tail. No. of calves—3, aborted once. Date of last parturition—August, 1925. The oestral periods have been at normal intervals. The cow has been served several times without result. Has been treated for metritis and infectious vaginitis. Contagious abortion test—positive. Tuberculin test—negative.	Vulva shows numerous dormant nodules on its mucosa. The vagina contains a small quantity of turbid tenacious mucus. Cervix is normal in size, very firm in consistency. Mucosa pale-pink. It is not possible to pass a small dilator through the ostium uterinum. Uterus mostly abdominal. Left horn somewhat larger than the right. Consistency appears normal. The left fallopian tube is enlarged and tortuous. Its fimbriated extremity is attached to the ovary, but is not cystic. Right enlarged and tortuous; its fimbriated extremity encapsules the ovary and is slightly cystic. The right ovary is the size of a small hen egg; firm in consistency. Left ovary the size of a pigeon's egg; firm in consistency. There are some fibrous attachments between the ovaries and the mesosalpinx. Broad ligaments normal. *Diagnosis*:—chronic catarrhal metritis, hydrosalpinx, pavilionitis.	Vulva shows many nodules in the mucosa. The vagina contains a small quantity of tenacious opaque mucus. Cervix, 9 × 3·4 cm., width of wall, 1·5 cm; mucosa pale, consistency very firm. The third and fourth cervical folds appear to be indurated. The ostium uterinum is very tortuous and exceedingly small in the anterior half. Uterine body is 10 × 4 cm.; width of wall, 1·2 cm.; left horn 30 × 3 cm.; width, 1—·8cm.; right horn, 27 × 3 cm.; width of wall, ·8 cm. Serosa smooth, pinkish-yellow in colour. Consistency slightly flaccid. The mucosa is moist and slightly swollen. It is pinkish-grey in colour. The cotyledons have almost entirely disappeared. There are some small greyish-yellow depressed areas, oval in outline, 2–4 mm. in size, scattered irregularly over the mucosa of both horns. Both fallopian tubes are enlarged at their cornual extremity. Towards the ostium abdominale they appear normal in size. The swollen portion of the tube is tortuous, thin-walled, white in colour, and contains an opaque greyish fluid. The fimbriated extremity of both tubes is attached to the corresponding ovary by fibrous adhesions, the right ovary being completely encapsuled. Broad ligaments normal.	Left ovary, 3 × 2 × 2 cm. Its surface is attached to the free border of the fimbriated extremity of the fallopian tube, and there are fibrous adhesions between it and the mesosalpinx. On section. the ovarian tissue is greyish-white in colour. It is very firm and shows the presence of four atrophied corpora lutea. There is a small cyst-like structure close to the surface of the ovary. Apparently a normal Graafian follicle. Right ovary, 4 × 3·6 × 3 cm. It is completely encapsuled by the fimbriated extremity of the fallopian tube. There are some loose fibrous adhesions between the inner aspect of the ovarian pocket and the surface of the ovary. Its posterior pole shows a small cyst over which the capsule is transparent. Its anterior pole contains a large corpus luteum which projects about 2 mm. above the surface of the ovary. On section. there is a large corpus luteum present 2·6 × 1·9 cm. It presents three small cystic excavations in its interior. Towards the opposite pole there are three small oval cysts, and a trace of lutein tissue.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Thymus.*—Small calcified area, otherwise normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Lymphocytic and eosinophilic infiltration in interacinous tissue. *Adrenals.*—Eosinophilic infiltration, irregular distribution of cell's resembling megakaryocytes in the cortex. *Vagina.*—round cell and neutrophilic infiltration with local degeneration and desquamation of epithelium. *Cervix.*—Cervicitis chronica catarrhalis with sclerosis. *Uterus.*—Endometritis catarrhalis chronica. *Ovaries.*— Right.—Atretic follicles, vascular dilatation, oedema. cystadenoma, corpus luteum normal. Left.—Sclerosis, atretic follicle. *Fallopian tubes.*—Hydrosalpinx, pavilionitis.	Class considered hopeless. No. treatment tried.	Metritis catarrhalis chronica, hydrosalpinx, pavilionitis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·3 × ·6 × ·6 cm..........	Weight 0·3 gm.	Supernumerary, 1·2 × ·8 × ·3 cm......	weight 0·5 gm.
Hypophysis, 2·3 × 1·7 × 1·4 cm........	,, 3·0 gms.	Thyroid—Left half, 7 × 3·4 × ·8 cm.	
Adrenals—Left, 4 × 5·8 × 1·5 cm.......	,, 17·5 ,,	Right half, 6·2 × 4·2 × ·8 cm.	
Right, 5·8 × 4·2 × 1·4 cm...	,, 19·5 ,,	Length, including isthmus, 29·8 cm., weight 22 gms.	

CASE 42. (See Appendix II, Fig. 41.)

FRIESLAND COW 2113, 17 MONTHS OLD (AT FIRST EXAMINATION).

Date of first examination, 8.7.26. *Date of slaughter*, 11.11.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 580 lb. Normal in appearance: udder lactating: mammary tissue normal. There is no history of the heifer having been served previous to examination. At the age of 13 months the udder increased in size and secreted normal milk. While under observation for 16 months the periods of oestrum were irregular with exceedingly long interoestral periods. Contagious abortion test, positive. Tuberculin test, negative.	Vulva shows lesions of granular vaginitis; it does not admit the hand. Vagina from the rectum appears normal. Cervix normal in size and consistency. Uterus and uterine horns somewhat large and walls slightly flabby. Reaction to palpation is slow. Both fallopian tubes are enlarged and tortuous; both pavilions form a small soft cyst, which is in close contact with the ovary. Right ovary size of a small hen egg, firm in consistency. Left ovary size of a golf-ball, rounded and fluctuating. Broad ligaments normal. *Diagnosis*:—metritis chronica catarrhalis, hydrosalpinx, pavilionitis. While under observation the right ovary became cystic.	Vulva and vagina normal. Cervix, 7 × 5 cm.; width of wall, 2·5 cm.; mucosa pale; there is a small quantity of opaque mucus. Uterus—body, 8 × 5 cm.; width of wall, 1 cm.; right and left horns, 18 × 4 cm.: width of wall, 1–1·2 cm. The serous surface is adherent by loose fibrous connections to the rectum and broad ligaments. On section, the mucosa is moist; there is complete absence of cotyledons; it is yellowish-grey in colour. There is a small quantity of opaque mucus in the left horn and brownish mucus in the right horn. Both fallopian tubes and their pavilions are enlarged and contain an opaque watery fluid.	Right ovary, 5 × 3·5 × 4·5 cm., is rounded and fluctuating; it is adherent through fibrous connections to the mesosalpinx, the uterine horn, and the broad ligament. On section, it contains a large cyst with a band of lutein tissue in its wall. There is little ovarian tissue remaining. Left ovary, 4 × 3·5 × 3·5 cm., is rounded and fluctuating: it also shows fibrous adhesions to the broad ligaments, the mesosalpinx, and the uterine horn. On section, it contains a large cyst with a thin band of lutein tissue in its wall. There is little ovarian tissue remaining.	*Epiphysis*.—Corpora amylacea. *Hypophysis*.—Normal. *Thymus*.—Normal. *Pancreas*.—Normal. *Adrenals*.— Right.—Normal. Left.—Normal. *Thyroid*.—Normal. *Supramammary lymph gland*.—Normal. *Mammary gland*.—Concretions. *Vagina*.—Normal. *Cervix*.—Normal. *Uterus*.—Metritis chronica catarrhalis. *Fallopian tubes*.— Right and Left.—Hydrosalpinx. *Ovary*.— Right.—Lutein cyst. Left.—Lutein cyst.	Case considered hopeless. No treatment tried. The cow was kept under observation to notice the oestral periods.	Metritis chronica catarrhalis, hydrosalpinx, pavilionitis, cystic oophoritis.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·5 × ·6 × ·6 cm.	Weight 0·4 gm.	Thyroid—Right, 7·5 × 5 × 1 cm.
Hypophysis, 2 × 1·8 × 1·3 cm.	„ 2·5 gms.	Left, 6 × 3·2 × 1 cm.
Adrenals—Right, 5·5 × 4 × 1·4 cm.	„ 12·5 „	Length, including isthmus, 20 cm., weight 25·5 gms.
Left, 4 × 3·3 × 1·5 cm.	„ 9·0 „	

CASE 43. (See Appendix II, Fig. 42.)

FRIESLAND COW, 9 YEARS OLD.

Date of first examination, 4.8.26. *Date of slaughter*, 24.8.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition poor. Weight 860 lb. There is slight relaxation of the pelvic ligaments with a depression on either side of the sacrum. There is deformity of the hoofs due to chronic laminitis, such as one sees in styfsiekte (phosphate deficiency) in cattle. The cow has lactated, but is now dry. The mammary tissue is soft and pliable. No. of calves. 5. Date of last parturition, December, 1925. Since last parturition, has been served several times without result. The cow was treated by the owner for infectious vaginitis. While under observation here, she showed two normal oestral periods. The previous history of her oestral periods is not known. Contagious abortion test, positive. Tuberculin test, positive.	The vulva shows lesions of infectious vaginitis in a subacute stage. The vagina contains a small quantity of thick tenacious mucus with a few yellowish flocculi. The cervix is ragged and the first cervical fold ectropic. Mucosa is pale pink. Os is patent. From the rectum the posterior third of the cervix is enlarged and firm. Length normal. Uterus and uterine horns are enlarged and flaccid. The uterus is mostly abdominal. It is lacking in tone. Right fallopian tube towards its anterior extremity is enlarged and firm. size of a lead pencil. Its fimbriated extremity is closed and forms a small firm enlargement, the size of a walnut. Left fallopian tube is enlarged and fluctuating. Its fimbriated extremity is enlarged to the size of a hen's egg, and forms a soft thin-walled cyst. Right ovary is difficult to isolate on account of surrounding adhesions It is the size of a walnut and firm in consistency. The left ovary is the size of a small hen egg. It also is difficult to isolate on account of fibrous adhesions. It appears to be somewhat fluctuating. Broad ligaments are adherent in places to the uterus and horns through loose fibrous adhesions. *Diagnosis*:—cervicitis. chronic catarrhal-metritis, hydrosalpinx, pavilionitis.	Vulva shows some lesions of infectious vaginitis in a subacute stage. The vagina contains a small quantity of tenacious mucus. Cervix, 9 × 6 cm.; width of wall. 3 cm.; mucosa pale pink in colour. The first cervical fold is ectropic and somewhat ragged. Uterus—colour. The first cervical fold is ectropic and somewhat ragged. Uterus—body, 11 × 6 cm.: width of wall, 1 cm.; left horn, 23 × 4 cm.; width of wall, 1 to 1·2 cm.; right horn, 25 × 3 cm.; width of wall, ·8 to 1·5 cm. The uterus is soft and flabby. The serosa is yellowish-pink in colour. There are numerous loose fibrous filaments, through which the uterus and uterine horns are adherent to the broad ligaments, the rectum, and the bladder. The mucosa is greyish-yellow and moist. It appears to be slightly swollen. It is entirely changed. There is no evidence of normal cotyledons. The mucosa is smooth except for occasional depressions, a couple of millimetres in diameter. The mucosa of the body shows two small patches, 1·5 cm. in diameter, dark brownish-grey to bluish-grey in colour, which extend a few millimetres into the mucosa, but do not reach the muscular coat. Right fallopian tube is enlarged and fluctuating in its anterior 2/3. The posterior 1/3 is normal in size. Its pavilion is adherent to the ovary by its free border and forms a large soft thin-walled cyst. Left fallopian tube is enlarged and firm in its anterior 2/3. Its posterior 1/3 is normal in size. Its fimbriated extremity forms a firm enlargement about the size of a walnut. Fibrous adhesions pass between both tubes and the corresponding ovary. The broad ligaments show fibrous attachments to the uterus and the uterine horns. P-m. showed tubercular lesions in the thoracic lymphatic glands.	Left ovary, 4·6 × 3 × 2·8 cm. The whole ovary is covered in a fibrous capsule through which its surface cannot be examined. On section, shows two cystic structures with lutein tissue in their walls. The largest is 1·7 cm. in diameter. It is a corpus luteum with a central cyst 1·2 cm. in diameter: around the cyst is a very narrow greyish zone an a band of lutein tissue 0·25 cm. in thickness. The contents of the cyst are thin. liquid, transparent, and slightly yellowish in colour. The second cyst is smaller and elongated, 1·1 cm. in length. Its wall is smooth and shows traces of lutein tissue. It contains a clear yellowish liquid. The cystic structures are surrounded by a thin capsule of greyish-white ovarian tissue. Right ovary, 4 × 2 × 2 cm. This ovary is for the most part also covered with a fibrous capsule through which its surface cannot be examined. On section the ovary is firm. greyish-white in colour. It shows several small cyst-like structures up to ·4 cm. in diameter, apparently normal Graafian follicles. It also shows traces of atrophied corpora lutea and a corpus albicans ·7 cm. in diameter. The ovary has not recently ovulated.	*Adrenals*.—Normal. *Thymus*.—Normal. *Pancreas*.—Normal. *Thyroid*.—Normal. *Epiphysis*.—Normal. *Hypophysis*.— Pars nervosa—some small concretions. Pars distalis—two vesicles lined with flattened epithelium. Both filled with colloid, which shows some calcareous deposit. *Supramammary lymph gland*.—fibrosis. *Mammary gland*.—Concretions, many neutrophiles in acini. *Vagina*.—Local round cell infiltration and epithelial desquamation. *Cervix*.—Normal.	Case considered hopeless. No treatment tried.	Metritis chronica catarrhalis, hydrosalpinx, pavilionitis. corpus luteum cysts.
					Uterus.—Slight changes in mucosa—oedema, capillary walls slightly thickened, slight round-cell infiltration. *Fallopian tubes*.—Hydrosalpinx. *Ovary*.— Right.—Atretic follicles, oedema, area showing large number of blood vessels cut transversely. In this area there is considerable fat. Left.—Two cysts with lutein tissue in the wall, one considerable thickness of lutein tissue, other shows traces only.	

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·3 × ·4 × ·3 cm.	Weight 0·15 gm.	Thyroid—Right half, 6 × 3·7 × ·9 cm.
Hypophysis, 2·4 × 1·4 × 1·5 cm.	,, 2·75 gms.	Left half, 6·4 × 4·5 × 1 cm.
Adrenals—Right, 5·8 × 4·2 × ·8 cm.	,, 15·7 ,,	Length, including isthmus, 20·9 cm., weight 37 gms.
Left, 4·7 × 3·7 × 1·5 cm.	,, 12·35 ,,	

CASE 44. (See Appendix II, Fig. 43.)

FRIESLAND COW 2115, 6 YEARS OLD.

Date of first examination, 1.8.26. *Date of slaughter*, 9.12.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 950 lb. Normal in appearance. Udder in the later stages of lactation. Udder tissue normal. No. of calves—2, including one abortion. Date of last parturition, August, 1925. Served several times without result. Oestral periods irregular with abnormally long inter-ovulation periods. Contagious abortion test, positive. Tuberculin test, negative.	Vulva shows a few enlarged lymph follicles. Mucosa pale pink. Vagina slightly stenosed so that the hand is admitted with difficulty. It contains a slight quantity of opaque mucus. Cervix mucosa normal; size and consistency normal. Uterus—body normal in size. Right horn much larger than the left. Wall of the right horn is firm and thickened. There are some loose fibrous adhesions between the uterus and broad ligaments. Both fallopian tubes are enlarged, but are difficult to palpate on account of fibrous tissue adhesions between the mesosalpinx, the uterus, and the ovary. The pavilion of both tubes is enlarged and cystic. Right ovary size of a pigeon's egg, firm in consistency; the pavilion of the tube attached. Left ovary size of a pigeon's egg, firm in consistency: the pavilion of the tubes attached. Broad ligaments show some fibrous adhesions to the uterus. *Diagnosis*: — endometritis chronica catarrhalis, perimetritis, hydrosalpinx, pavilionitis. While under observation the left ovary became cystic.	Vulva unchanged. Vagina shows a slight stenosis, as the result of a scar on its dorsal wall. Cervix, 7 × 6 cm.; width of wall, 2·8 cm.; mucosa normal. Uterus—body, 14 × 6 cm.; width of wall, 1·5 cm.; left horn, 19 × 4 cm.; width of wall, ·8 cm.; right horn, 20 × 4·5 cm.; width of wall, 1·2 cm. Serous surface of the uterus shows some loose fibrous adhesions between its dorsal aspect, the broad ligaments, and the rectum. Right horn is somewhat larger than the left. On section, the mucosa is pale pinkish-grey in colour, swollen and moist. The cotyledons have almost entirely disappeared, their positions being represented by a small oval depression, varying in size from 4 millimetres to 2 millimetres. Both tubes are enlarged and tortuous. Their pavilions are adherent to the corresponding ovary and are cystic. They contain a slightly opaque liquid. The tubes are surrounded by fibrous tissue adhesions, which connect the mesosalpinx to the ovary and the uterine horn.	Left, 5 × 4 × 4 cm. Its surface shows numerous fibrous adhesions through which it is adherent to the pavilion of the tube and the mesosalpinx. It is rounded and appears to contain a large cyst. On section, there are two cysts, one is 2 cm. in diameter, rounded, and shows in its wall a capsule of lutein tissue, up to 4 millimetres in thickness. Lining is smooth, greyish in colour. The second cyst is halfmoon-shaped, 3 cm. in diameter. Its capsule is thin. Its lining is greyish and smooth. Little ovarian tissue is left; it is greyish-white in colour and somewhat moist. Right, 3 × 2 × 2·5 cm. Shows fibrous adhesions similar to the left. It is firm in consistency. On section, there is a normal Graafian follicle ·75 cm. in diameter. There is an atrophied corpus luteum ·4 cm. in diameter. The remainder of the ovarian tissue is greyish-white in colour and firm in consistency.	*Thymus gland*.—Normal. *Pancreas*.—Normal. *Adrenal*.—Right and Left: Normal. *Epiphysis*.—Normal. *Hypophysis*.—Marked hyperaemia of the pars distalis. *Supramammary lymph gland*.—Normal. *Mammary gland*.—Concretions. *Vagina*.—Normal. *Cervix*.—Normal. *Uterus*.—Metritis chronica catarrhalis. *Fallopian tubes*.—Salpingitis, pavilionitis. *Ovary*.— Left.—Cystic corpus luteum, small cyst without lutein tissue in its wall, Graafian follicle cyst, oedema, fibrous adhesions. Right.—Atretic follicles, oedema, fibrous filaments.	No treatment tried. Case considered hopeless.	Endometritis chronica catarrhalis, hydrosalpinx, pavilionitis, cystic oophoritis (corpus luteum and Graafian follicle cyst in the left ovary).

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis. 1·4 × ·4 × ·4 cm.	Weight 0·3 gm.	Thyroid—Right half, 5·2 × 5 × 1·2 cm.
Hypophysis, 2·5 × 1·5 × 1·2 cm.	,, 3·0 gms.	Left half, 5 × 3·8 × ·8 cm.
Adrenals—Right, 5 × 3·2 × 1·2 cm.	,, 12·0 ,,	Length, including isthmus, 19 cm., weight 22·5 gms.
Left, 6 × 4·2 × 1·2 cm.	,, 13·0 ,,	

CASE 45. (See Appendix II, Fig. 44.)

SHORTHORN COW No. 2141, 4 YEARS OLD.

Date of first examination, 22.8.26. *Date of slaughter*, 26.10.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 900 lb. Normal in appearance. The distribution of fat over the body is irregular, such as one sees in a functionally sterile female. The heifer has never calved. While under observation for a period of two months at this institute, did not show oestrum. The owner, however, reported that she had been served several times without result. Tuberculin test.—Negative Contagious abortion test.—Suspicious; agglutination 1–50.	Vulva small, admits three fingers with difficulty. From the rectum the vagina appears normal. The cervix is difficult to palpate. It appears elongated, dilated, and elastic on palpation. The uterus presents only the right horn, which is somewhat enlarged and contains fluid. The left horn cannot be palpated. Both fallopian tubes appear normal, but the uterine extremity of the left tube cannot be traced to an attachment with the uterine horn. Right ovary oval in shape, firm in consistency. Left ovary oval in shape, slightly larger than the right ovary, firm in consistency. Broad ligaments normal. *Diagnosis*: deformity of the genital tract, uterus unicornus, stenosis of the cervix.	The vulva is normal; there is a constriction in the region of the hymen which admits only two fingers The vagina is normal Cervix—10 × 6 cm; width of wall over the annular folds is 2 cm, between the annual folds ·5 cm The cervix is very much enlarged, soft, and elastic On section, it shows three compartments which are separated by the annular folds The lumen of the cervical canal, through the annual folds, is exceedingly small, about 2 mm. The compartments are filled with material similar to the cervical plug of pregnancy, but less tenacious The condition is a false uterine seal The uterus presents only one horn, namely, the right, 24 × 4 cm It is markedly tapering towards its cornual extremity The serosa is smooth. On section, it contains a thick tenacious brownish-orange coloured material. Mucosa pale greyish-white in colour and smooth There is no evidence of cotyledons present Left horn is absent. One can trace the remnants of Mueller's duct. The right fallopian tube is normal. Left fallopian tube ends in a small cystic *cul-de-sac* towards its uterine extremity.	Right ovary, 3·5 × 2 × 1·2 cm. It is firm in consistency and shows numerous Graafian follicles. On section, the ovary tissue is greyish-pink in colour and firm in consistency. There are numerous small developing follicles. No trace of lutein tissue is present. Left ovary, 4 × 2·3 × 2 cm. Shows numerous Graafian follicles, the largest of which is 1·3 cm. in diameter. There is also a large corpus luteum, which is on a level with the surface of the ovary. On section, the corpus luteum of the last interovulation period fills up most of the ovary, size 1·5 cm. It is brownish-orange in colour. There is a small central greyish area Close to the corpus luteum is a follicle 1·3 cm in diameter. There are numerous small developing follicles.	*Epiphysis.*—Corpora amylacea. *Hypophysis.*—Pars distalis: hyperaemia, vesicle containing colloid. *Pancreas.*—Normal. *Adrenals.*—Right and left: Normal. *Thymus.*—Hassal's corpuscles very numerous in medulla, some very large with faintly staining nuclei. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Parenchymatous tissue badly developed, mostly fibrous tissue and fat, occasional mammary acini. *Fallopian tubes.*—Villi somewhat thicker than normal. *Cervix.*—Epithelial cells charged with mucus. They are somewhat swollen. *Ovary.*— Left.—Corpus luteum with much fibrous stroma, normal follicles. some hyheraemia and oedema. Right.—Normal follicles, atretic follicles, hyperaemia, and oedema. *Uterus.*—Mucosa shows some atrophy.	Case considered hopeless. No treatment tried.	Cervical deformity with stenosis of the ostium uterinum. This caused retention of oestral debris. The original lesion was followed by corpus luteum persistens and the development of a false uterine plug. Further deformity, not associated with sterilty, consisted in uterus unicornis and maldevelopment of the left fallopian tube.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·4 × ·6 × ·6 cm.	Weight 0·3 gm.	Thyroid—Left, 4 × 3·5 × 1 cm.
Hypophysis, 1·8 × 1·6 × 1·4 cm.	,, 3·0 gms.	Right, 3·8 × 2·5 × ·8 cm.
Adrenals—Left, 3·2 × 2·8 × 1·5 cm.	,, 6·5 ,,	Length, including isthmus, 18·8 cm., weight 14·5 gms.
Right, 5·5 × 3 × 1·4 cm.	,, 9·0 gms.	

CASE 46. (See Appendix II, Fig. 45.)

SHORTHORN COW No. 2142, 3½ YEARS OLD.

Date of first examination, 22.8.26. *Date of slaughter*, 2.11.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 780 lb. Normal in appearance. Has never lactated. Has never shown oestrum. There is no evidence that this animal was was the result of twin conception. Contagious abortion test, negative. Tuberculin test, negative.	Vulva admits three fingers with difficulty, mucosa normal; anteriorly it ends in a *cul-de-sac*. Through the rectum no cervix and no vagina can be felt. The genital tract cannot be traced in front of the *cul-de-sac* of the vagina. Two small bodies, probably ovaries, can be felt in the anterior part of the broad ligaments. These are, however, small, size of a pea, and undeveloped. The broad ligaments are thin and short. *Diagnosis*: arrest in development of the genital organs. (Freemartinism).	Vulva admits three fingers only. Mucosa pale; length, 10 cm. It ends in a gradually decreasing *sal-de-sac* 3 cm. in front of the meatus urinarius. On either side of the apex of the *cul-de-sac* there are two small openings, which admit a probe for a distance of 4 m. These openings also end blindly. Stretching forward on either side from the *cul-de-sac* there are 2 short remnants of Wolffian ducts, that on the left 4 cm., that on the right 5 cm. long. The ducts of Mueller can be traced forward between a dorsal and ventral serous covering. They have not united, but show occasional cystic dilations along their length. The anterior extremity of Mueller,s ducts cannot be traced towards the undeveloped ovary. The broad ligaments are extremely thin and undeveloped.	Left ovary, 1·2 × ·6 × ·4 cm. It is covered with a fairly well-marked fatty capsule. It is firm in consistency. On section, the ovarian tissue is uniformly brownish-red in colour, and firm in consistency. It is embedded in fatty tissue. It does not appear to have ovulated. Right ovary, 1 × ·5 × ·4 cm. It is covered with a fairly well-marked fatty capsule and is firm in consistency. On section, the ovarian tissue is brownish-red in colour and is firm in consistency. It does not appear to have ovulated.	*Epiphysis.*—Normal. *Hypophysis.*—Some vesicles filled with colloid in pars distalis. *Thymus.*—No change. *Thyroid.*—No change. *Pancreas.*—No change. *Adrenals.*—Right and left: there appears to be some thickening of the fibrous capsule. The fibrous strands in the zona glomerulosa appear to be somewhat thickened. The medulla is very rich in nerves. *Supramammary lymph gland.*—No change. *Mammary gland.*—Very badly developed, mostly composed of fatty tissue in which can be recognized isolated groups of glandular acini. *Ovary.*—Few small cysts lined with connective tissue, one primary follicle seen. No trace of lutein tissue, or corpus albicans.	No treatment.	Arrest in the development of the genital organs ("Freemartinism.").

CASE 47. (See Appendix II, Fig. 46.)

FRIESLAND COW 2143, 13 YEARS OLD.

Date of first examination, 1.8.26. *Date of slaughter*, 5.10.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility
		Genital Tract.	Ovaries.			
Condition good. Weight 1,100 lb. Normal in appearance. Udder has lactated, but is now dry. Udder tissue normal. The cow has had several calves. Last parturition—17·9.20. While under observation for two months, there was no evidence of oestrum. Contagious abortion test.—Positive. Tuberculin test.—Negative.	Vulvar mucosa normal. There is a small prominent cyst, the size of a pigeon's egg, in the right gland of Bartholini. Vagina contains a small quantity of somewhat tenacious mucus. Cervical mucosa normal. There is a small quantity of mucus, which is somewhat tenacious, in the ostium uterinum. Per rectum, the cervix is slightly enlarged and firm. The uterus and uterine horns appear normal in size. In the dorsa-lateral wall of the pars indivisa, there is a firm oval enlargement about the size of a small hen egg; similar but smaller enlargements are felt in both horns. Some small enlargements, about the size of marbles, can be felt throughout the uterine walls. The larger swellings appear on careful palpation to contain fluid. Both fallopian tubes normal. Right ovary the size of a pigeon's egg, oval in shape, firm in consistency. Surface is somewhat irregular, with two small prominences. Left ovary somewhat smaller in size, firm in consistency. Broad ligaments are normal. *Diagnosis*: endometritis chronica catarrhalis cystica.	Vulva shows a cyst in the right gland of Bartholini. Mucosa normal. Vagina contains a small quantity of clear, tenacious mucus. Cervix, 9 × 5 cm.; width of wall, 2·4 cm. mucosa pale yellowish-pink. Ostium uterinum is patent, contains a small quantity of clear, slightly tenacious mucus. Uterus—body, 10 × 5 cm.; width of wall, 1·4 cm.; left horn, 20 × 3 cm.; width of wall, 1 cm.; right horn, 22 × 3 cm.; width of wall, 1·2 cm. Serosa smooth, yellowish-pink in colour. On section, the mucosa is pale yellowish-grey to pinkish-grey in colour; there is a small quantity of mucus present. Cotyledons are not prominent; they are yellowish-grey in colour. The wall of the pars indivisa shows a large, fluctuating ovoid enlargement 4 × 3·4 × 2 cm.: smaller enlargements of a similar nature are found in both horns, the smallest being about ·8 cm. in diameter. These enlargements project above the mucosa, and are covered with a transparent capsule. On section they are smooth-walled and contain an opaque fluid with some yellowish-grey flocculi. The smaller ones are confined to the mucosa, but the larger extend somewhat into the muscular coat. Nine of these cysts are present, two in the pars indivisa, seven scattered over both horns. Fallopian tubes normal. There is a slight fibrous adhesion between the mesosalpinx and the left ovary. Broad ligaments normal.	Left ovary, 2·6 × 1·8 × 1·8 cm. Firm in consistency. Its surface shows traces of seven atrophied corpora lutea, the largest being 3 mm. in diameter. There are several cyst-like structures, up to 4 mm. in diameter, apparent through the surface. These are apparently normal follicles. On section, the ovarian tissue is greyish-yellow in colour and firm in consistency. There is a superficial small corpus luteum (2) and several small follicles. There is no large follicle evident. Right ovary. 3·5 × 2·5 × 2 cm. There is a corpus luteum present, which stands fairly prominently above the surface. It is ·7 cm. in diameter. Traces of six atrophied corpora lutea are also present. There are several apparently normal Graafian follicles. On section, there are several small follicles, and the corpus luteum of the last interovalation period, which is ·7 × 1 cm.	*Epiphysis.*— Corpora amylacea, arteriosclerosis in the surrounding vessels. *Hypophysis.*— Vesicle in pars distalis. Local degeneration of cells in isolated acini, due to pressure from new fibrous tissue (sclerosis). *Thyroid.*—Normal. *Thymus.*—Local accumulation of neutrophiles around Hassal's corpuscles. *Adrenals.*—Normal. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Fallopian tubes.*—Normal. *Uterus.*—Endometritis chronica cattarrhalis cystica. *Ovaries.*—Corpus luteum persistens, shows increase in fibrous tissue, normal follicles in both ovaries.	Case considered hopeless. No treatment tried. Cow slaughtered for examination.	Endometritis chronica catarrhalis with cystic degeneration of the mucous glands, corpus luteum persistens.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·4 × 1·8 × 1·3 cm.	Weight 3·5 gms.
Epiphysis, 1·3 × 0·4 × 0·4 cm.	,, 0·2 gm.
Adrenals—Right, 4 × 3 × 1·3 cm.	,, 9·0 gms.
Left, 4·6 × 3 × 1·2 cm.	,, 10·0 ..

Thyroid—Right. 5·3 × 4·6 × 1·3 cm.
Left. 6·5 × 5 × 1 cm.
Length, including isthmus, 26 cm., weight 32 gms.

CASE 48. (See Appendix II, Fig. 47.)

FRIESLAND COW 2146, 10 YEARS OLD.

Date of first examination, 17.9.26. *Date of slaughter*, 9.11.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,370 lb. Normal in appearance. Has lactated, but is now dry. Udder tissue normal. The cow has had several calves. Date of last parturition, 25 5 21 (abortion). Since last parturition oestral periods have been irregular with long interovulation periods. Tuberculin test, negative. Contagious abortion test, positive.	Vulva normal. Vagina contains a quantity of thick tenacious mucus. Cervix very ragged First cervical fold ectropic. The ostium uterinum admits the finger to a depth of 5 cm. Per rectum, the cervix is enlarged and firm. Uterus mostly abdominal. Left horn larger than the right, flaccid. The wall appears to be somewhat thickened. Fallopian tubes normal. Right ovary is the size of a small hen egg, elongated, contains the corpus luteum of the last interovulation period. Left ovary size of a small hen egg, elongated, firm in consistency, contains an apparently normal follicel. Broad ligaments are unchanged. *Diagnosis:* cervicitis chronica, endometritis chronica catarrhalis.	Vulva and vagina are normal. Cervix, 9 × 6 cm.; width of wall, 3 cm. Mucosa pale pink. First cervical fold ectropic. It presents numerous small polypus-like growths. The second fold is also hypertrophied and shows some polypus-like growths. The ostium uterinum is dilated throughout, so that it admits one finger easily. Uterus—body, 11 × 6 cm.; width of wall, 1·2–1·4 cm.; right horn, 20 × 4 cm.; width of wall, 1 cm.; left horn, 23 × 5 cm.; width of wall, 1·3 cm. Serosa smooth yellowish-pink. On palpation appears somewhat soft. The mucosa is greyish-pink in colour. In the left horn there are whitish-yellow yellow elevations representing the cotyledons. In the right horn the mucosa is smooth, somewhat transparent, with no trace of cotyledons. Both fallopian tubes are normal. Broad ligaments normal.	Left ovary, 4·4 × 3 × 2·2 cm. Contains a corpus luteum of the last interovulation period, also some traces of atrophied corpora lutea. There are several small subcapsular follicles. On section, the corpus luteum is of a pinkish-brown colour and 2·1 cm. in diameter. Several apparently normal follicles are also seen. The ovary tissue is greyish-pink in colour and firm in consistency. Right ovary, 3·6 × 2 × 1·8 cm. It contains a large ripe follicle and several small subcapsular follicles. There are traces of several atrophia corpora lutea. On section, there are several apparently normal follicles and traces of atrophied corpora lutea. The ovary tissue is greyish pink in colour and firm in consistency.	*Thymus.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Adrenals.*—Normal. *Hypophysis.*—Normal. *Epiphysis.*—Normal. *Supramammary lymph gland.*—Fibrosis. *Mammary gland.*—Fibrosis. *Vagina.*—Normal. *Cervix.*—Fibrosis with some polypus formation. *Uterus.*—Metritis chronica catarrhalis, with some hyaline degeneration of the capillaries in the mucosa. *Fallopian tubes.*—Normal. *Ovaries.*— Left.—Corpus luteum—normal, normal follicles. Right.—Normal follicles.	No treatment tried. Cow slaughtered for examination.	Cervicitis chronica, endometritis chronica catarrhalis.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·8 × 1·7 × 1·4 cm.	Weight 3·5 gms.
Epiphysis, 1·2 × 0·7 × 0·7 cm.	,, 0·3 gm.
Adrenals—Left, 4·5 × 4 × 2 cm.	,, 19·5 gms.
Right, 6·5 × 4 × 1·8 cm.	,, 20·0 ,,

Thyroid—Left, 6 × 5 × 1·2 cm.
Right, 6·8 × 5 × 1·3 cm.
Length, including isthmus, 22 cm., weight 38 gms.

CASE 49. (See Appendix II, Fig. 48.)

FRIESLAND COW 2148, BORN 23.4.18.

Date of first examination, 22.8.26. *Date of slaughter*, 24.8.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition obese. Weight 1,920 lb. The general appearance is coarse and somewhat ox-like; there is a very irregular distribution of fat; large lumps of fat are prominent on the buttocks, hips, and dorsal region. The udder has lactated, but is now dry. Date of last parturition, 31/10/22; since parturition, oestrum has been irregular and infrequent. While under observation at this Institution for a period of 12 months, oestrum appeared in Sept., 1926, Feb. and June, 1927; the only oestral periods observed. Contagious abortion test, positive. Tuberculin test, negative.	The animal was first examined during an oestral period; the genital tract showed the typical condition associated with oestrum. The left uterine horn was somewhat larger than the right, otherwise the uterus appeared normal; both fallopian tubes and broad ligaments normal. The right ovary contained an ovulation fossa. Left ovary: size of a small hen egg and normal in consistency. *Diagnosis*: genital tract appears normal (functional sterility due to obesity).	Vulva normal. Vagina contains a very small quantity of clear mucus. Cervix, 9 × 5 cm.; width of wall, 2·5 cm. Mucosa pale; ostium uterinum contains a small quantity of very tough opaque mucus; it is very sinuous, and difficult to penetrate. Uterus—wall, 10 × 6 cm.; width of wall, 1·25–1·5 cm.; right horn, 20 × 3·5 cm.; width of wall, 1 cm.; left horn, 20 × 3 cm.: width of wall, 1 cm. Serosa normal. On section, the cavum uteri contains a small quantity of tenacious opaque mucus. The mucosa of the right horn is greyish to greyish-pink in colour, smooth, and somewhat moist; the cotyledons are not apparent, having apparently undergone atrophy. The left horn is dark greyish-red in colour. The cotyledons are prominent yellowish-red in colour. Both fallopian tubes are unchanged. Both broad ligaments are unchanged.	Right ovary, 4·2 × 3 × 2 cm. It contains the corpus luteum of the last interovulation period; there is a cyst-like prominence 1·5 cm. diameter upon its convex border (apparently a developing follicle). On section, there is a corpus luteum, which is embedded, 2 cm. in diameter, of a brown-orange colour. A few apparently normal developing follicles are present. The ovarian tissue is whitish-grey in colour. Left ovary, 4 × 2·8 × 2·2 cm. Corpus luteum (II) is present, as a small yellowish-brown speck on its surface. There are several apparently normal follicles seen through the surface. On section, there are several areas of lutein tissue, the remains of previous corpora lutea. Several apparently normal developing follicles are present. The ovarian tissue is firm and greyish-white in colour.	*Epiphysis.*—Normal. *Hypophysis.*—Several small vesicles containing colloid in pars intermedia. *Pancreas.*—Normal. *Thyroid.*—Normal. *Thymus.*—Normal. *Adrenals.*—Local hyaline degeneration of cortex. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vulva.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Slight atrophy of the mucosa. Some enlarged mucous glands. *Fallopian tubes.*—Normal. *Ovaries.*— (1) Normal follicles, small cyst lined with epithelium, atretic follicles. (2) Normal corpus luteum, normal follicles.	No treatment tried. Animal used as a control.	Functional sterility resulting from obesity, followed by degeneration of the uterine mucosa.

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·6 × 0·75 × 0·75 cm....... Weight 0·75 gm.
Hypophysis, 2·5 × 2·5 × 1·5 cm........ ,, 4·0 gms.
Adrenals—Left, 6·5 × 3·5 × 1·5 cm..... ,, 21·0 ,,
Right, 5 × 4·5 × 1·5 cm..... ,, 21·5 ,,

Thyroid—Right, 5·8 × 8 × 1·5 cm.
Left, 7 × 6·5 × 1 cm.
Length, including isthmus, 25·5 cm., weight 46·5 gms.
Parathyroid, 1·5 × 0·5 cm.

CASE 50. (See Appendix II, Fig. 49.)

FRIESLAND COW No. 2149, AGE 14 YEARS.

Date of first examination, 22.8.26. *Date of slaughter*, 19.10.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1.200 lb. Normal in appearance. The udder has lactated, but is now dry. Udder tissue normal. The cow has had several calves, which included two abortions. Last parturition, 11/4/23. Since last parturition, the oestral periods have been irregular, with long interovulation periods. Tuberculin test, negative. Contagious abortion test, positive.	Vulva normal. Vagina contains a small quantity of tough, tenacious opaque mucus. Cervix is very ragged; first cervical fold ectropic and hypertrophied; second cervical fold can be seen through the ostium uterinum externum; it is also hypertrophied. Mucosa pale pink. The ostium uterinum admits the forefinger throughout its entire length. Per rectum, the cervix towards its caudal extremity is markedly enlarged. The uterus and uterine horns are enlarged, flaccid, and lacking in tone. Right ovary size of a small hen egg, firm in consistency. Left ovary size of a small hen egg, firm in consistency. Broad ligaments are normal. *Diagnosis* cervicitis catarrhalis chronica, with induration and polypus formation.	Vulva and vagina are normal. Cervix, 10 × 7 cm.; width of wall, 3 cm. Mucosa pale pink. First cervical fold is markedly cetrocpic and ragged. It shows two polypus-like enlargements hanging from the central part of the cervical fold. The second fold is also markedly hypertrophied. There is a small cyst 4 mm. in diameter, which stands out prominently on its ventral surface. The wall appears to be abnormally firm. Uterus—body, 11 × 6 cm.; width of wall, 1·4 cm.; both horns equal in size, 20 × 3·5 cm.; width of wall, 1·5 cm. Serosa smooth yellowish-pink. Mucosa greyish-pink in colour, moist, and somewhat transparent. Cotyledons fairly prominent and of a greyish-white colour. Both fallopian tubes are normal. Broad ligaments normal.	Right ovary, 4 × 2·4 × 2·4 cm. Surface smooth, firm in consistency; eight atrophied corpora lutea are apparent through the surface, as well as several apparently normal Graafian follicles. On section, the ovarian tissue is pinkish-grey and firm. It shows the presence of several small cyst-like structures, apparently normal Graafian follicles. Left ovary, 4 × 3 × 2·7 cm. Surface smooth, firm in consistency. There are six atrophied corpora lutea present, as well as several apparently normal follicles. The largest of the latter is 1·5 cm. in diameter. The corpus luteum of the last interovulation period is extremely large, 3·5 × 2·5 × 2·5 cm. It stands prominently above the ovary. Last ovulation took place 12 days previous to slaughter. On section, the ovarian tissue is pinkish-grey and firm. Traces of several atrophied corpora lutea are present. The large corpus luteum already described presents a halfmoon-shaped appearance on section. There are many small cyst-like structures, apparently normal follicles.	*Adrenals.*— Right.—Normal. Left.—Normal. *Epiphysis.*—Normal. *Hypophysis.*—Pars distalis very rich in blood-vessels. *Thyroid gland.*—Normal. *Thymus.*—Normal. *Pancreas.*—Insterstitial tissue somewhat thickened. *Supramammary lymph gland.*—Normal. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Polypus and induration. *Uterus.*—Metritis chronica catarrhalis. *Fallopian tubes.*—Normal. *Ovary.*—Left and Right: Several atretic follicles and small cystic degeneration and oedema. Few normal follicles. Normal corpus luteum.	No treatment tried. Cow slaughtered for examination.	Cervicitis chronica catarrhalis, with polypus formation. Small cystic degeneration of the ovaries, metritis chronica catarrhalis (both the latter conditions were demonstrated on microscopic examination).

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1 × 0·5 × 0·5 cm.	Weight 0·2 gm.
Hypophysis, 2·8 × 2·6 × 1·6 cm.	„ 8·5 gms.
Adrenals—Left, 6·7 × 4 × 2 cm.	„ 23·5 „
Right, 5 × 5·2 × 2·2 cm.	„ 20·0 „

Thyroid—Right, 8 × 7 × 1 cm.
Left, 8 × 7·5 × 1·5 cm.
Length, including isthmus, 30·5 cm., weight 53·5 gms.

CASE 51. (See Appendix II, Fig. 50.)

FRIESLAND COW 2150, AGE 15 YEARS.

Date of first examination, 22.8.26. *Date of slaughter*, 12.10.26.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,100 lb. Normal in appearance. Has lactated, but is now dry. Udder tissue normal. Has had several calves, which included two abortions. Last parturition, 9/9/19. Oestral periods irregular. Interovulation period long. Tuberculin test, negative. Contagious abortion test, positive.	Vulva and vagina normal. Cervix ragged, first cervical fold ectropic. Mucosa normal. From the rectum it seems abnormally firm. Uterus appears to be somewhat larger than normal, but of good tone. The uterine horns appear similarly enlarged, but also of good tone. Fallopian tubes normal. Right ovary size of a small hen egg and appears cystic. Left ovary size of a pigeon's egg, firm in consistency. There is a small cyst-like area, apparently a Graafian follicle. Broad ligaments normal. *Diagnosis* :—induration of the cervix, corpus luteum cyst.	Vulva and vagina normal. Cervix, 8 × 4·5 cm.; width of wall, 2·5 cm. Mucosa pale pink. The posterior fold is ectropic and markedly ragged. It shows several small polypus-like enlargements. The second and third cervical folds are also somewhat hypertrophied. Uterus—body, 11 × 4·5 cm.; width of wall, 1 cm.; left horn, 20 × 3·5 cm.; width of wall, 1·5–1 cm.; right horn, 19 × 3·5 cm.; width of wall, 1·8–1·3 cm. Serosa pale yellowish-pink. Mucosa greyish-red to greyish-blue in colour, smooth, moist, transparent. Some greyish-yellow areas, 1·5–2 cm. in length, can be seen through the mucosa, apparently the remains of cotyledons. The wall of both horns appears somewhat thickened and cuts rather more firm than normal. Both fallopian tubes normal. Broad ligaments normal. The left half of throid gland showed a few enlarged vesicles containing amber-coloured colloid.	Right ovary, 4 × 3 × 3 cm., oval in shape, fluctuating. It shows several traces of atrophied corpora lutea. On section, there is a cyst 2·5 cm. in length, which contains an amber-coloured fluid. The wall is smooth and yellowish-pink in colour. Outside the immediate lining of the cyst a very thin capsule of lutein tissue can be recognized. Towards the anterior pole there are two small cyst-like structures, 1 cm. in diameter, apparently normal follicles. There is a thick band of ovarian tissue remaining, which is pinkish-grey to white in colour. Left ovary, 4 × 2·5 × 2 cm. Firm in consistency; it contains a large prominent corpus luteum. This has developed since the last ovulation period, 17 days previously. The surface of the ovary shows several traces of atrophied corpora lutea. On section, there is a large dark orange-coloured corpus luteum 2·6 × 2·5 × 2 cm. There are some normal follicles. The ovarian tissue is pinkish-grey and firm in consistency	*Epiphysis.*—Corpora amylacea and arteriosclerosis. *Hypophysis.*—Vesicle formation and sclerosis of senility. *Adrenals.*—Marked sclerosis, isolation of tissue islets by connective tissue. Fatty degeneration of isolated islets. *Thymus.*—Increase in the medullary tissue with decrease in cortical tissue. *Pancreas.*—Normal. *Supramammary lymph gland.*—Normal. *Thyroid.*—Struma colloides. *Mammary gland.*—Induration, concretions. *Cervix.*—Induration. *Uterus.*—Endometritis chronica catarrhalis. *Fallopian tubes.*—Normal. *Ovaries.*—Cystic oophoritis, small cystic degeneration with epithelial lining, adenoma, atretic follicles, lutein cyst. Normal follicles also present.	No treatment tried. Cow slaughtered for examination.	Corpus luteum cyst. Endometritis chronica catarrhalis was evident microscopically.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·5 × 1·8 × 1·8 cm.	Weight	5·0 gms.
Epiphysis, 1·4 × 0·8 × 0·7 cm.	,,	0·35 ,,
Adrenals—Left 5·3 × 4 × 1·5 cm.	,,	16·5 ,,
Right, 6·7 × 3·3 × 1·6 cm.	,,	18·0 ,,

Thyroid—Left, 5·5 × 5 × 1 cm.
Right, 5·5 × 4·6 × 1·3 cm.
Length, including isthmus, 20 cm., weight 26·5 gms.

CASE 52. (See Appendix II, Fig 51.)

FRIESLAND COW No. 2160, AGE 11 YEARS.

Date of first examination, 27.8.26. *Date of slaughter*, 7.9.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,340 lb. Appearance normal. The left anterior portion of the udder is atrophied and indurated; the three remaining quarters are non-lactating and normal in consistency. No. of calves—3. Last parturition — July, 1923. The oestral periods since last parturition have been irregular, for the most part, with interovulation periods varying from four to six weeks; sometimes two months elapsed between oestral periods. The last oestral period occurred eight days prior to slaughter. It had occurred without ovulation apparently. There was never any symptom of nymphomania. Tuberculin test and contagious abortion test negative.	Vulva normal. Vagina contains a small quantity of tenacious mucus. Cervix-mucosa pale, first cervical fold ectropic and ragged, ostium uterinum patent. Uterus and uterine horns normal in size and consistency; on palpation both horns react readily and become firm. Fallopian tubes—right is enlarged, somewhat tortuous, and fluctuating: its ovarian extremity ends in a fluctuating enlargement about 1½ cm. in diameter; it is adherent to the ovary; left fallopian tube is normal in size. Right ovary size of a small hen egg; it contains the corpus luteum of the last interovulation period. It is softer and more flabby than normal. Left ovary somewhat smaller than the right, it contains a developing follicle. The broad ligaments are normal. *Diagnosis*:—hydrosalpinx on the right side, chronic cervicitis.	Vulva normal. Vagina mucosa normal, contains a quantity of opaque tenacious mucus. Cervix, 9 × 6 cm.; width of wall, 3 cm. Mucosa pale; first cervical fold is ectropic and ragged, the ostium uterinum contains a small quantity of tenacious mucus. Uterus body, 9×5 cm.; width of wall, 1 cm.; left horn 16 × 3 cm.; width of wall, 1 cm.; right horn, 17 × 3 cm.; width of wall, ·8 cm. Serosa normal; mucosa is greyish-pink in colour, smooth, and somewhat moist; cotyledons of the left horn are greyish-red in colour and prominent; cotyledons of the right horn are small or entirely absent towards the apex of the horn; they are dull yellowish-red in colour. Left fallopian tube is normal in size and consistency. Right fallopian tube is thickened throughout and for the most part fluctuating and thin-walled; it contains an opaque liquid; its fimbriated extremity is adherent to the ovary, and is cystic. The broad ligaments are unchanged.	Left ovary, 3 × 2 × 2 cm.; shows some traces of lutein tissue and several apparently normal follicles on its surface. On section, there is a cyst-like structure, apparently a normal follicle, 1·5 cm. in diameter. There are several smaller follicles. Traces of the corpora lutea of former interovulation periods are present. The ovarian tissue is greyish-white and firm. Right ovary, 5 × 5 × 4·8 cm.; is rounded and fluctuating throughout; its surface shows fibrous adhesions, to which it is adherent to the mesosalpinx. On section, there is a large cyst with a capsule varying from 2 mm. to 5 mm. The immediate lining is yellowish in colour. The cyst has a capsule of lutein tissue: outside this capsule there is a narrow band of greyish-white ovarian tissue which remains.	*Epiphysis.*—Normal. *Hypophysis.*—Normal. *Thymus.*—Medullary areas increased in size. *Pancreas.*—Normal. *Adrenals.*—Normal. *Thyroid.*—One enlarged vesicle, with secondary vesicles developed from the epithelium. *Mammary gland.*—Induration, mastitis catarrhalis. *Cervix.*—Cervicitis catarrhalis. *Uterus.*—Atrophy of mucosa with diminution of the uterine glands. Some glands enlarged and cystic. *Fallopian tubes.*— Right.—Hydrosalpinx. Left.—Normal. *Ovary.*— Right.—Corpus luteum cyst. Left.—Small cyst (Graafian follicle type); some vessels show hyaline degeneration.	No treatment tried. Cow retained for observation.	Hydrosalpinx, lutein cyst, degeneration of the uterine mucosa, with commencing cyst formation in the uterine glands (microscopic).

SIZE AND MEASUREMENTS OF ENDOCRINES.

Epiphysis, 1·3 × 0·4 × 0·4 cm.	Weight 2·0 gms.
Hypophysis, 2·7 × 1·7 × 1·5 cm.	,, 3·75,,
Adrenals—Left, 6 × 3 × 1·8 cm.	,, 12·5 ,,
Right, 6 × 3·8 × 1·4 cm.	,, 14·5 ,,

Thyroid—Right, 6 × 6 × 1 cm.
Left, 7 × 3·5 × 1 cm.
Length, including isthmus, 23 cm., weight 30·5 gms.

CASE 53. (See Appendix II, Fig. 52.)

FRIESLAND COW 2161, AGE 8 YEARS.

Date of first examination, 27.8.26. *Date of slaughter*, 31.8.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition very fat. Weight 1.200 . The cow has been dehorned, but the head appears somewhat coarse; the neck is also slightly heavy. The udder has not lactated, there is no history of pregnancy. While under observation at this Institute, there was no oestral period between September, 1926, and March, 1927; between March and June, 1927, there were seven oestral periods, some of which were at intervals of two weeks, others of four weeks apart. In July and August there was no oestrum. Contagious abortion test and tuberculin test both negative.	Vulva small, does not admit the hand; mucosa pink. Vagina from the rectum appears normal. Cervix is slightly small, firm in consistency. Uterus and uterine horns are smaller than normal, somewhat soft and thin-walled. Both fallopian tues are normal. The right ovary is the size of a pigeon's egg, firm; it contains a prominent corpus luteum. The left ovary is somewhat smaller than the right and firm in consistency. No evidence of developing Graafian follicles, although the cow had not shown oestrum for some weeks previously. Broad ligaments normal. *Diagnosis*:—hypoplasia of the uterus (functional sterility).	Vulva and vagina normal. Cervix, 8 × 4 cm.; width of wall, 2 cm., firm in consistency; the ostium uterinum contains a small quantity of tenacious mucus. Uterus—body, 7 × 2·5 cm.; width of wall, ·6 cm.; horns, 14 × 2 cm.; width of wall, ·5 cm. It is somewhat flabby and thin-walled. Serosa normal; cavum uteri contains a small quantity of opaque greyish mucus; mucosa is greyish-pink in colour, smooth and moist; there is no evidence of the cotyledons of the non-pregant uterus. Both fallopian tubes are normal; both broad ligaments normal.	Left ovary, 3 × 2 × 1·6 cm.; shows traces of lutein tissue on its surface; several apparently normal Graafian follicles are apparent through the surface. On section, there are several small cyst-like structures, apparently normal Graafian follicles. The ovarian tissue is whitish-grey in colour, and somewhat moist. Right ovary, 3 × 2·5 × 3 cm., contains a corpus luteum of the last inter-ovulation period, which had already existed over two months. There are several apparently normal Graafian follicles apparent through the surface. On section, the corpus luteum is 2·5 cm. in diameter, and orange-yellow in colour. There are several apparently normal follicles. The ovarian tissue is greyish-white and somewhat moist.	*Epiphysis.*—Normal. *Hypophysis.*—Hyperaemia of pars distalis and intermedia. *Thyroid gland.*—Normal. *Pancreas.*—Normal. *Adrenals.* Fatty infiltration in zona glomerulosa, and some irregularity in the distribution of lipoid granules in the cortex. *Thymus.*—Local areas in medulla show accumulations of eosinophiles close to Hassal's corpuscles. *Suprammary lymph gland.*—Normal. *Mammary gland.*—Small isolated areas of parenchyma, mostly fibrous tissue and fat. *Cervix.*—Normal. *Uterus.*—Degeneration of the mucosa. Diminution in glands, some glands filled with desquamated epithelial cells, some increase in fibrous tissue. *Fallopian tubes.*—Normal. *Ovaries.*—Small cysts, oedema, atretic follicles. Hyaline degeneration of a few vessels. *Corpus luteum.* Normal.	This cow was kept as a control. No treatment tried.	Hypoplasia of the uterus, with degeneration of the mucosa. (May be the result of an old-standing chronic metritis or functional sterility.)

Size and Measurements of Endocrines.

Epiphysis, 0·5 × 1·25 × 0·5 cm........	Weight 0·4 gm.	Parathyroids (4).—
Hypophysis, 2 × 1·5 × 1·25 cm.........	,, 3·5 gms.	(1) 1·5 × 0·75 × 0·25 cm.
Adrenals—Right, 4·5 × 4 × 1·25 cm....	,, 12·5 ,,	(2) 1·2 × 0·75 × 0·2 cm.
Left, 6 × 3·5 × 1 cm.........	,, 14·0 ,,	(3) 0·75 × 0·6 × 0·2 cm.
Thyroid—Right, 5 × 4 × 1 cm.		(4) 0·7 × 0·7 × 0·3 cm.
Left, 5 × 4·5 × 1 cm.		
Length, including isthmus, 29·5 cm.......	,, 29·5 ,,	

CASE 54. (See Appendix II, Fig. 53.)

FRIESLAND COW 2162, AGE 5 YEARS.

Date of first examination, 27.8.26. *Date of slaughter*, 15.1.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition fair. Weight 870 lb. The appearance is normal. The udder has lactated. but is now dry. Mammary tissue normal. Number of calves—1 (a twin gestation), 29/7/24. Since parturition, the cow had been served several times without result. Oestral periods occurred every 24 days while under observation. Tuberculin test. negative. Contagious abortion test, negative.	The vulva is small and does not admit the hand. Per vagina, palpation not possible. The vagina from the rectum shows nothing unusual. Cervix is normal. The uterus is normal in tone and size. Left horn somewhat larger than the right. Horns lie in front of the pubic brim. There are a few adhesions on the dorsal aspect of both horns. Both fallopian tubes are enlarged, and the right is very tortuous. The fimbriated extremity of the right tube appears to be attached to the ovary, partly encapsuling it. Right ovary, size of a walnut : firm. It is difficult to palpate on account of its encapsulation by the extremity of the tube. but a corpus luteum can be felt. Left ovary. small, firm ; two apparently normal follicles present. Broad ligaments unchanged. *Diagnosis* :—hydrosalpinx. pavilionitis.	Vulvar mucosa pink. There are some isolated yellowish nodules. The vagina is normal. Cervix, 7 × 5 cm. ; width of wall, 2·5 cm., pale pinkish-white in colour. It is normal in conformation and consistency. Uterus—body, 10 × 5 cm. ; horns—right, 19 × 3 cm. ; left. 21 × 3 cm. ; width of wall. 1 cm. Serosa shows a few fibrous adhesions in the dorsal aspect of both horns. Mucosa is pale yellowish-grey in colour. The cotyledons are small and more or less level with the surrounding surface of the mucosa. They are yellowish-grey in colour. Both fallopian tubes are enlarged throughout the entire length. The right is markedly convoluted. Its extremity is attached to the ovary. which is partly encapsuled. and forms a small soft cyst. The contents of both tubes is greyish opaque, liquid, with some white flocculi. The fimbriated extremity of the left tube is not attached to this ovary by its free border, but within the funnel there are some fibrous filaments.	Right ovary, 3 × 2·5 × 2 cm. It is firm on palpation. Its surface cannot be seen, as it is partly encapsuled by the fimbriated extremity of the tube and fibrous adhesions which pass between it and the mesosalpinx. On section. there are two small structures (·5 cm.) which are cyst-like filled with fluid. probably normal follicles. A trace of an atrophied corpus luteum can be seen. The remainder of the ovarian tissue is greyish-yellow in colour and firm in consistency. Left ovary. 2·5 × 2·5 × 2 cm. There is a large corpus luteum. 1·2 cm. in diamter, which projects well above the surface of the ovary. It is pale yellowish-grey in colour. There is a central cavity, 3 mm. in diameter, which contains fluid. Surrounding this cavity there is a whitish-grey zone, 1–2 cm. in thickness. Close to the corpus luteum is a cyst-like structure ·8 cm. in diameter, which is filled with fluid. There is little ovarian tissue, just a narrow band, which is greyish-yellow in colour.	The endocrines were not examined microscopically. *Mammary gland* appears unchanged. *Supramammary lymphatic glands.*—Normal. *Vulvar mucosa.*—Normal. *Vagina.*—Normal. *Cervix.*—Normal. *Uterus.*—Increase in fibrous tissue in the mucosa. The epithelium of uterus and the uterine glands is unchanged. *Fallopian tubes.*—Hydrosalpinx. *Ovary.*— Right.—Normal. Left.—Corpus luteum, oedema, small cystic degeneration. atretic follicles.	No treatment tried. Case considered hopeless.	Metritis catarrhalis chronica (microscopic). hydrosalpinx, pavilionitis.

CASE 55. (See Appendix II, Fig. 54.)

FRIESLAND COW 2163, AGE 4 YEARS.

Date of first examination, 3.9.26. *Date of slaughter*, 21.9.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition poor, normal in appearance. Weight 770 lb. The udder has lactated, but is now dry; its tissue appears normal. No. of calves, 1. Date of parturition, May, 1926. The afterbirth was retained. Since last parturition, there was no evidence of oestrum, which did not appear during 12 months whilst the animal was under observation at this Institution. Contagious abortion test—negative. Tuberculin test—negative.	Tail and buttocks soiled with purulent exudate. Vulva admits the hand with difficulty; mucosa reddish: Vagina contains a quantity of liquid pus, which is foetid. The cervix is much enlarged, mucosa reddish; the ostium contains liquid pus; a probe passes easily. The uterus is much enlarged, especially the left horn, it appears firm and thick-walled; there are adhesions between the uterus and the pelvic organs so that accurate palpation is difficult. The fallopian tubes and ovaries cannot be palpated, as their position is the site of thick fibrous adhesions. The broad ligaments are not definitely palpable. The pelvic lymphatic glands are enlarged and firm. *Diagnosis.*—Pyometra with sclerosis of the uterine wall and cervix. While under observation the uterus increased in size, especially the left horn; the ostium uterinum became completely occluded. Pus accumulated in the left horn and the pars indivisa.	Vulva normal. Vagina contains a small quantity of muco-purulent fluid. The cervix 11 × 8 cm.; width of wall, 4 cm.; it is firm, first cervical fold ectropic and ragged; the cranial half of the ostium uterinum is entirely closed. Uterus—body, 13 × 12 cm.; width of wall, 3 cm.; left horn, 21 cm.; width of wall, 1–2 cm.; right horn, 23 × 8 cm.; width of wall, 3–5 cm. The whole surface is covered with thick fibrous adhesions through which it is adherent to the pelvic organs and the rumen; the mass is difficult to dissect out. On section, uterus contains 4 litres thin yellowish, very foetid fluid; mucosa is greyish-black in colour: the wall is exceedingly firm. Either fallopian tube cannot be traced; their position is the site of dense fibrous adhesions. The broad ligaments are attached to the fibrous mass. The pelvic lympathic glands are enormously enlarged, firm on section, and mottled-red to greyish-yellow in colour. Note.—The right lateral ventricle of the brain was distended with clear fluid.	Left ovary cannot be found; it has apparently been embedded in dense fibrous tissue. The right ovary is embedded in fibrous tissue; it is 3 × 2 × 2 cm. On section, it is greyish-white in colour, very firm in consistency; there is no macroscopic evidence of corpora lutea or Graafian follicles.	*Epiphysis.*—Corpora amylacea. *Hypophysis.*— Pars distalis—hyperaemia. Pars nervosa.—Increase in fibrous tissue. *Thyroid.*—Normal. *Thymus.*—Mostly medullary tissue, few Hassal's corpuscles, few clumps of neutrophiles in the medulla. *Adrenals.*— (1) Normal. (2) Normal. *Pancreas.*—Normal. *Parathyroid.*—Normal. *Supramammary lymph gland.*—Some fibrosis. *Mammary gland.*—Concretions, fibrosis. *Cervix.*—Sclerosis. *Uterus.*—Pyometra, fibrosis, abscess formation. *Ovary.*—Sclerosis, no normal tissue seen.	Case considered hopeless; no treatment tried. (The cow was kept under observation for 12 months to note the oestral periods.)	Pyometra with sclerosis of the cervix and uterus.

SIZE AND WEIGHTS OF ENDOCRINES.

Hypophysis, 2·4 × 1·7 × 1·3 cm.	Weight	4·0 gms.
Epiphysis, 1·25 × 0·7 × 0·7 cm.	,,	0·75 gm.
Adrenals—Right, 5 × 3·5 × 1·5 cm.	,,	16·0 gms.
Left, 5·5 × 3·5 × 1·7 cm.	,,	20·0 ,,

Thyroid—Right, 6 × 5 × ·6 cm.
Left, 5·5 × 3·5 × 0·7 cm.
Length, including isthmus, 23·5 cm., weight 28 gms.
Parathyroids, 0·9 × 0·6 × 0·3 cm.

CASE 56. (See Appendix II, Fig. 55.)

HEREFORD COW 2264, AGE 10 YEARS.

Date of first examination, 4.2.27. *Date of slaughter*, 11.2.27.

General Appearance and History.	Gross Examination.	Post-mortem Examination of the Genital Apparatus.		Microscopic Examination.	Treatment.	Cause of Sterility.
		Genital Tract.	Ovaries.			
Condition good. Weight 1,350 lb. Head is coarse and somewhat ox-like. Lumbar region is slightly hollow, especially at its junction with the sacrum. Root of tail is elevated and the pelvic ligaments are relaxed. Udder has lactated, but is now dry. Udder tissue is normal. Has had several calves. Last parturition, 6/8/24. Since last parturition, the oestral periods have been irregular with long intervals. Tuberculin test, negative. Contagious abortion test, positive.	Vulvar lips are enlarged and flabby. Mucosa normal. On the right side, there is a small cyst in the gland of Bartholini. Vagina contains a small amount of tenacious mucus, which is somewhat opaque. Cervix is ragged and the first cervical fold ectropic, with polypus formation. Mucosa reddish. The ostium uterinum admits the middle finger easily. The circumference of the cervix is enlarged, and it is very firm in consistency. Uterus body and horns are enlarged and flabby. Scattered throughout the wall one can feel, on careful palpation, cyst-like structures varying in size up to a pigeon's egg. Attached to the apex of the right horn is a large tumour, the size of a child's head, which is firm in consistency. It hangs over the pelvic brim and draws the horn forward. Both fallopian tubes are easily palpable. They appear normal in size. Both fimbriated extremities appear normal. Right ovary, normal in size. It contains a corpus luteum. The ovary appears normal in consistency. Left ovary, normal in size. There is an apparently normal well-developed follicle (?) present. The remainder of the ovary is firm in consistency. Right broad ligament is elongated. *Diagnosis*: metritis chronica catarrhalis cystica, new-growth, cervicitis.	Vulvar mucosa normal. There is a small cyst in the right duct Bartholini, which contains clear fluid. Vagina contains a small quantity of tenacious mucus and some greyish flocculi. Cervix—first cervical fold ectropic and ragged; mucosa shows some reddish spots; size, 11 × 8 cm.; width of wall, 3·5 cm. Ostium uterinum admits one finger easily. Uterus—body, 13 × 5 cm.; width of wall, 1·5 cm.; left horn, 23 × 3 cm.; width of wall, 1 cm.; right horn, 26 × 4 cm.; width of wall, 1·5 cm. Serosa pinkish-yellow in colour. In the right horn of the uterus, apparently in its lateral wall, there is a firm tumour 17 × 14 × 11 cm. The tumour is covered with smooth serosa. On section, the uterus contains 10 c.c. of opaque greyish-fluid with flocculi; mucosa is greyish to slate colour. Scattered throughout the mucosa are numerous cyst-like elevations, varying in size up to 2·5 cm. in diameter. The cysts appear to be confined to the mucosa; they are smooth-walled and contain opaque greyish fluid. The right horn of the uterus is patent at the site of the tumour, which has developed in its wall. Both fallopian tubes are 2 mm. in thickness. They appear normal on palpation. Tumour, on section, cuts firm and fibrous. It is uniformally dense throughout. It is pale greyish-white in colour. It is easily enucleated. It is covered for the most part with serosa from the surface of the uterus. Over its median aspect there is a small covering of mucosa, where it encroaches on the mucosa uterinae.	Left, 5 × 3 × 2 cm. Firm in consistency, shows a large apparently normal follicle (?) and several smaller follicles on its surface. On section, there are two apparently normal follicles present. The ovariantissue is greyish-pink and firm. Right, 4 × 2·8 × 2· cm., firm in consistency. The corpus luteum of the last interovulation period is present and several small follicles are visible through the surface. On section, there is a small bright yellow corpus luteum of the last interovulation period. It has a whitish centre. There is an apparently normal follicle. The ovarian tissue is greyish-pink and firm in consistency.	*Adrenals.*—Normal. *Epiphysis.*—Normal. *Hypophysis.*—Normal. *Thymus.*—Normal. *Thyroid.*—Normal. *Pancreas.*—Normal. *Supramammary lymph gland.*—Fibrosis. *Mammary gland.*—Normal. *Vagina.*—Normal. *Cervix.*—Some fibrosis. *Uterus.*—Metritis chronica catarrhalis cystica. (One abscess seen in a mucous gland. The periphery was rich in neutrophiles, the centre had undergone degeneration with inspissation and calcification.) New-growth uterus—myofibroma. *Ovary.*— Right.—Some follicles showing commencing cystic degneration. The ovum showing degeneration is present in one follicle. Left.—Shows similar degenerative changes in the follicles. *Fallopian tubes.*—Normal.	Case hopeless. No treatment tried.	Metritis chronica catarrhalis, cystica, Cervicitis. Newgrowth uterine wall (myofibroma).

SIZE AND WEIGHTS OF ENDOCRINES.

Epiphysis, 1·4 × 0·4 × 0·4 cm.	Weight 0·3 gm.
Hypophysis, 2·5 × 1·5 × 1·2 cm.	,, 3·0 gms.
Adrenals—Left, 6 × 4·2 × 1·2 cm.	,, 13·0 ,,
Right, 5 × 3·2 × 1·2 cm.	,, 12·0 ,,

Thyroid—Left, 5 × 3·8 × 0·8 cm.
Right, 5·2 × 5 × 1·2 cm.
Length, including isthmus, 19 cm., weight 22·5 gms.

CASE 57. (See Appendix II, Fig. 56.)

Genital organs of heifer 579.

Uterus shows tubercular lesions in the mucous membrane.

The fallopian tubes and the ovaries are free.

Tubercular lesions were also present in the lungs, bronchial, mediastinal, and retropharyngeal lymphatic glands.

The ovaries are in an inactive state. There are several Graafian follicles in a static state. There is no recent corpus luteum. The corpus luteum of the last interovulation period is very small and chocolate in colour. It is just visible on the surface of the ovary as a brown speck about 2 mm. in diameter.

CASE 58. (See Appendix II, Fig. 57.)

Shows static state of the ovaries of a cow which suffered from functional sterility because of obesity.

CASE 59. (See Appendix II, Fig. 58.)

Portion of the vagina, cervix, and uterus of a cow showing an acquired stenosis of the cervix and uterus bipartitus completus.

CASE 60. (See Appendix II, Fig. 59.)

Bovine; genital tract sent from Durban Abattoir; specimen 6048.

The right ovary, 3.5 × 2.4 × 2.5 cm., is oval in shape and firm. Its surface is smooth, of a pinkish-grey to yellow colour. It shows four irregular areas of lutein tissue on its surface. Towards its anterior pole there is a prominence over which the capsule is thin, showing a yellow colour underneath. On section, practically the whole ovary is made up of yellow, crumbly, slightly adhesive material showing admixture with white hair. The hairs are not very numerous, but they are diffusely dispersed throughout. The tissue is easily enucleated from the capsule, from which several hairs appear to grow. The inner surface of the capsule is otherwise smooth. Towards the posterior pole of the ovary, the capsule is up to a thickness of .6 cm.; it appears greyish-white in colour, but does not show macroscopic evidence of developing Graafian follicles. That the ovary has ovulated would appear evident from the presence of the remains of corpora lutea. The left ovary, 2 × 1.3 × 1.4 cm. shows a prominent corpus luteum of a pale orange colour. On section, the corpus luteum fills practically the entire ovary. The ovary tissue remaining is represented by a narrow band around the apparently normal corpus luteum.

CASE 61. (See Appendix II, Fig. 60.)

Portion of the genitalia of a cow, showing a fibroma in the dorsal wall of the pars indivisa of the uterus.

CASE 62. (See Appendix II, Fig. 61.)

Portion of the genitalia of a cow, showing cervical deformity. Fig. b′ shows a small central cavity in corpus luteum 1.

CASES 63 AND 64.

Shows the changes in the rump seen in cows suffering from cystic degeneration of the Graafian follicle and nymphomania.

APPENDIX II.

In this appendix are shown photographs of the genitalia of cows slaughtered and also the photographs of a few individual cows which are considered interesting or in which the lesions producing sterility caused anatomical changes in the general appearance.

The photographs were taken by Mr. Theo. Meyer, of this Institution, from coloured drawings made of the fresh genitalia by Miss Letty and Miss Boezaart, artists at the Institution.

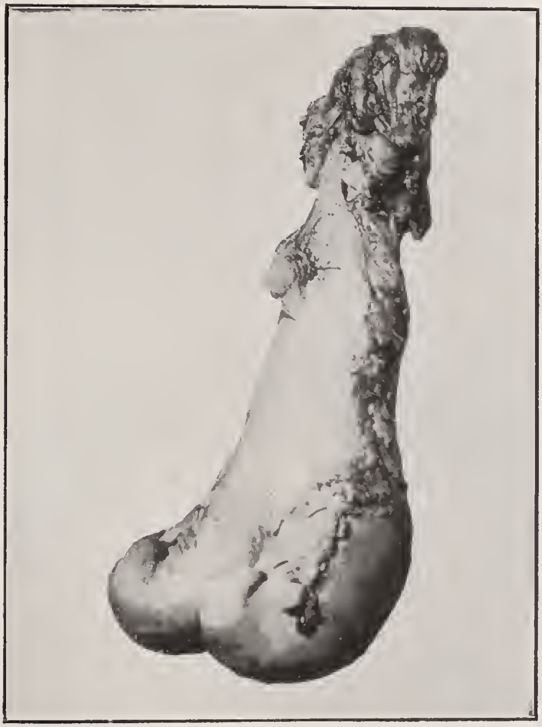
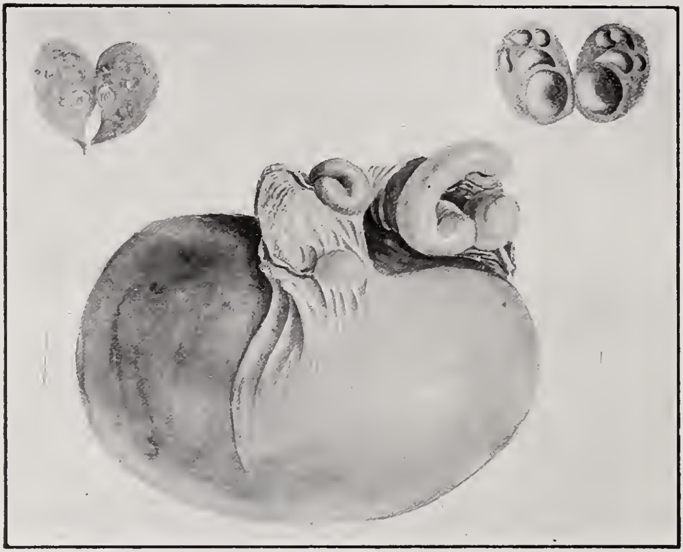


Fig. 1. (Case 1, App. I.)

A.



B.

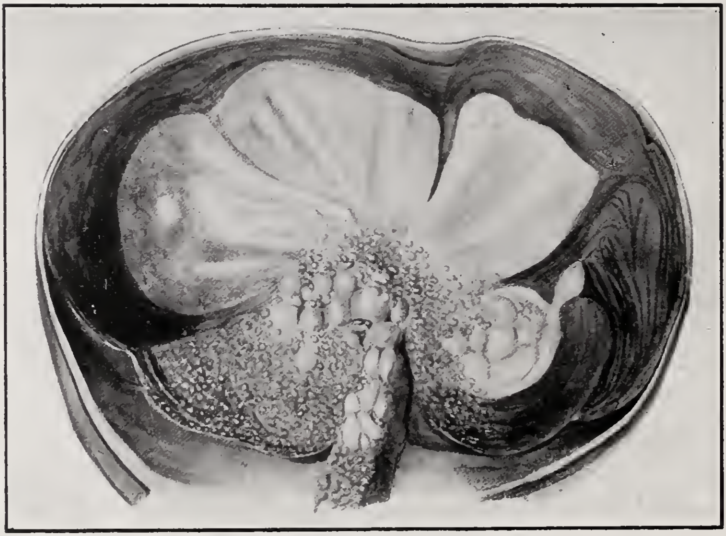


FIG. 2. (Case 2, App. I.)

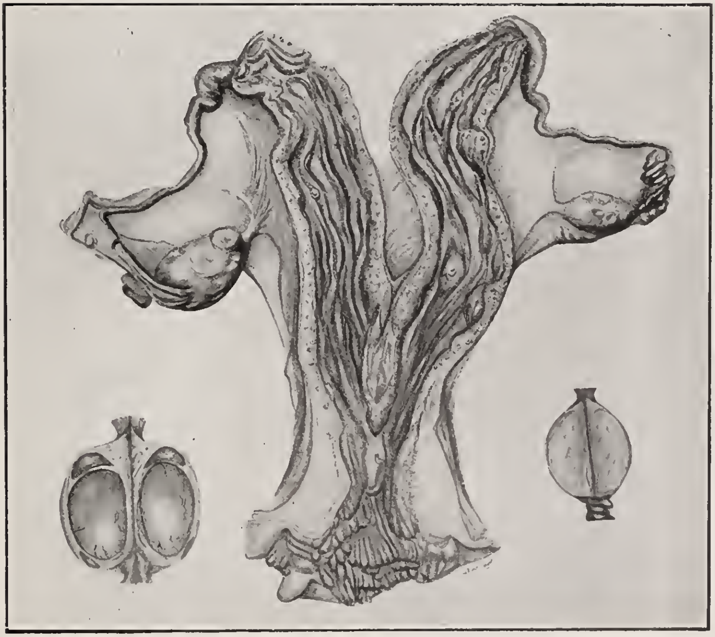


FIG. 3. (Case 3, App. I.)

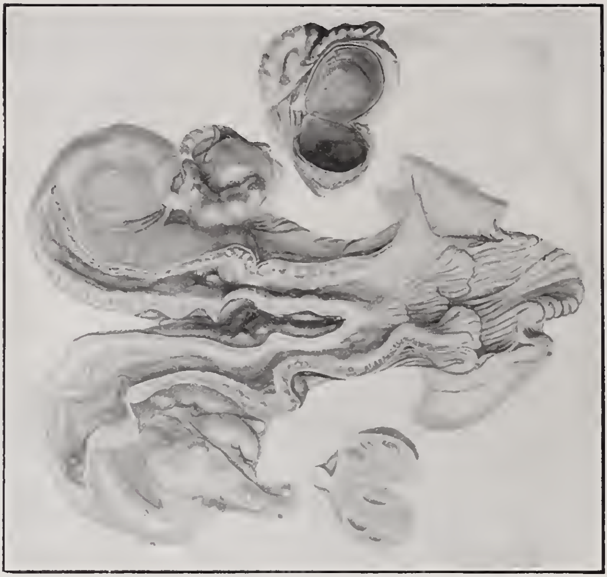


Fig. 5. (Case 5, App. I.)

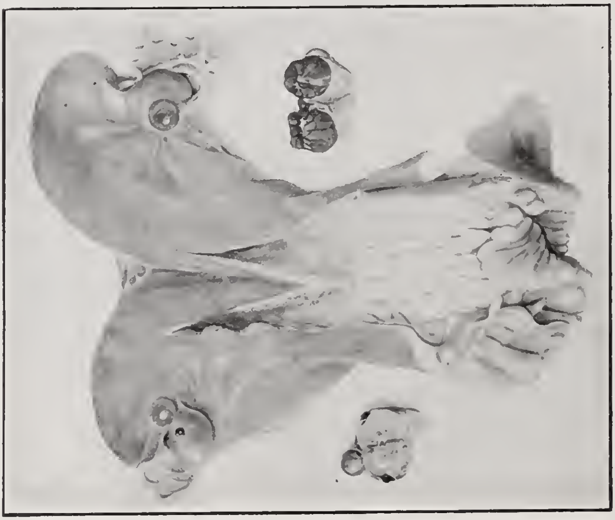


Fig. 4. (Case 4, App. I.)

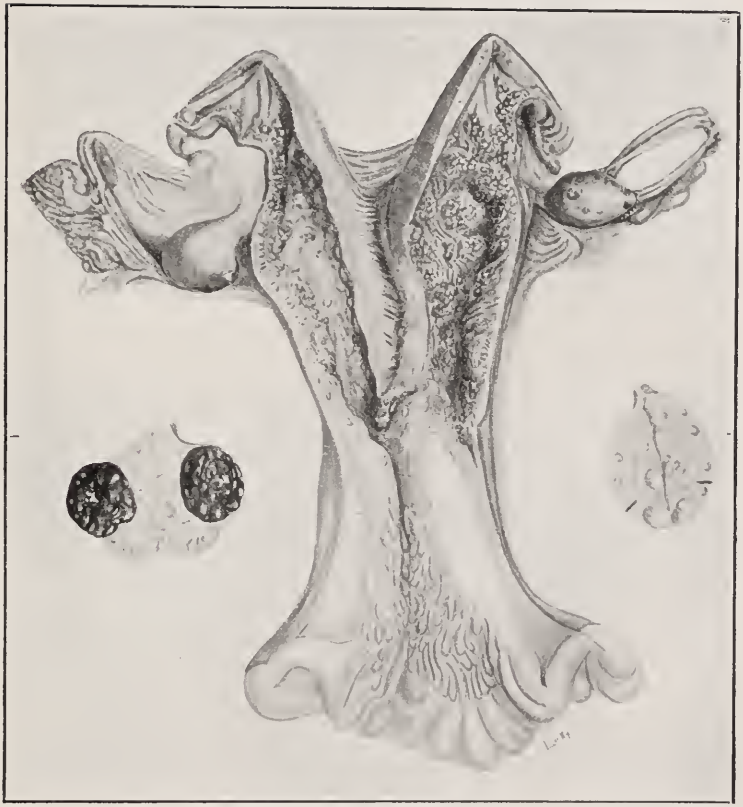


Fig. 6. (Case 6, App. I.)

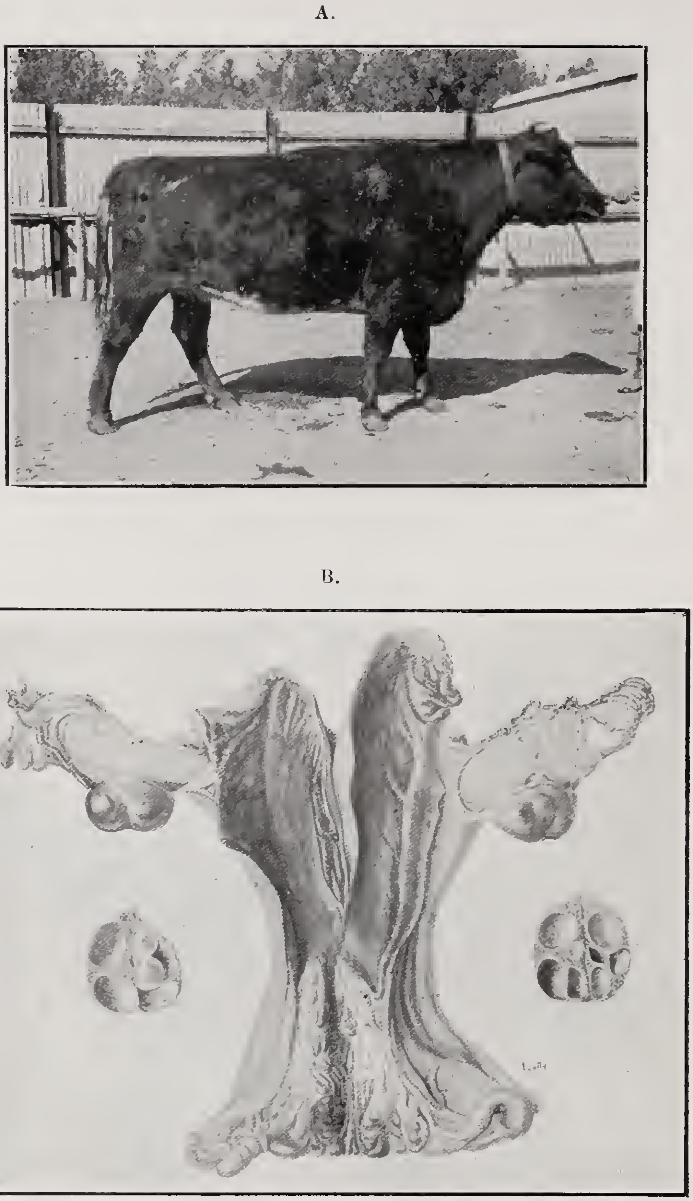
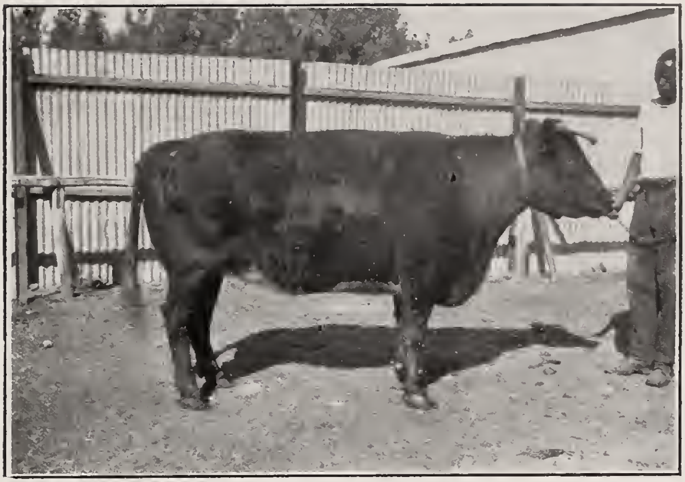


Fig. 7. (Case 7, App. I.)

A.



B.

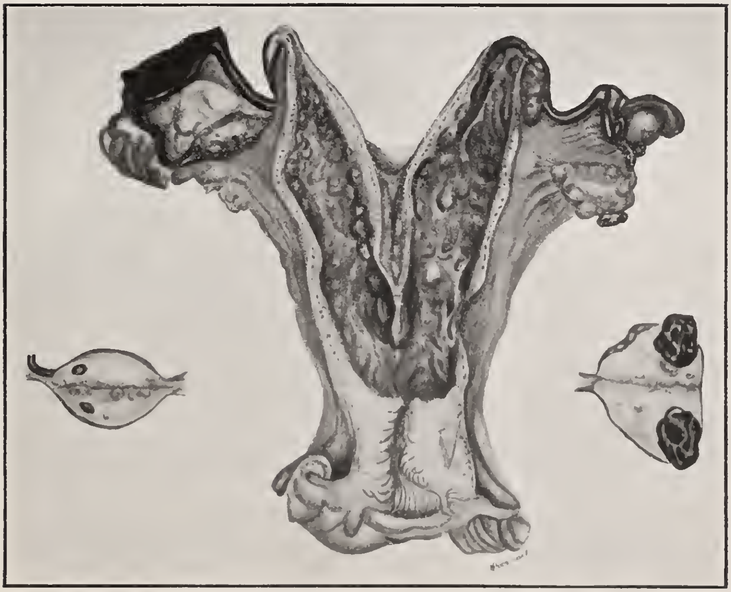


Fig. 8. (Case 8, App. I.)

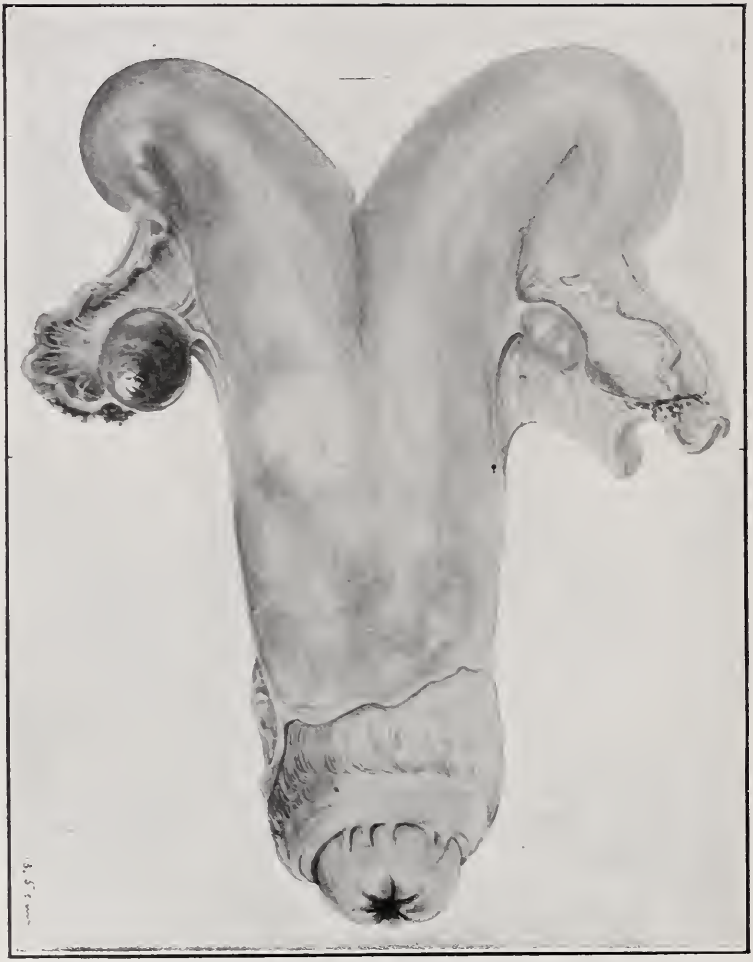
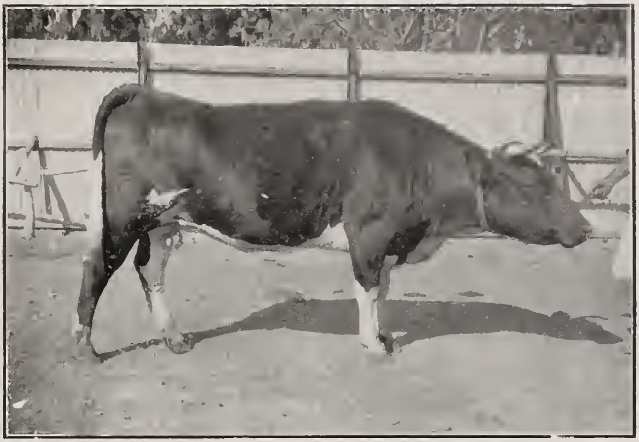


Fig. 9. (Case 9, App. I.)

A.



B.

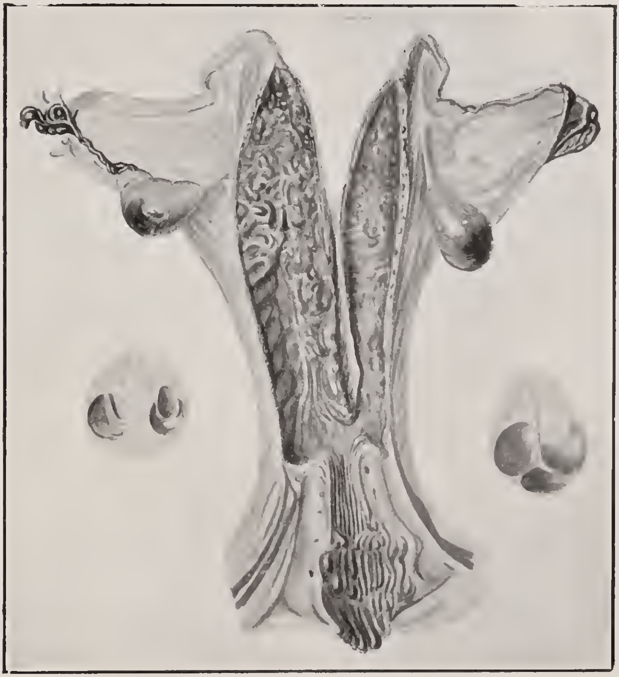


Fig. 10. (Case 10, App. I.)

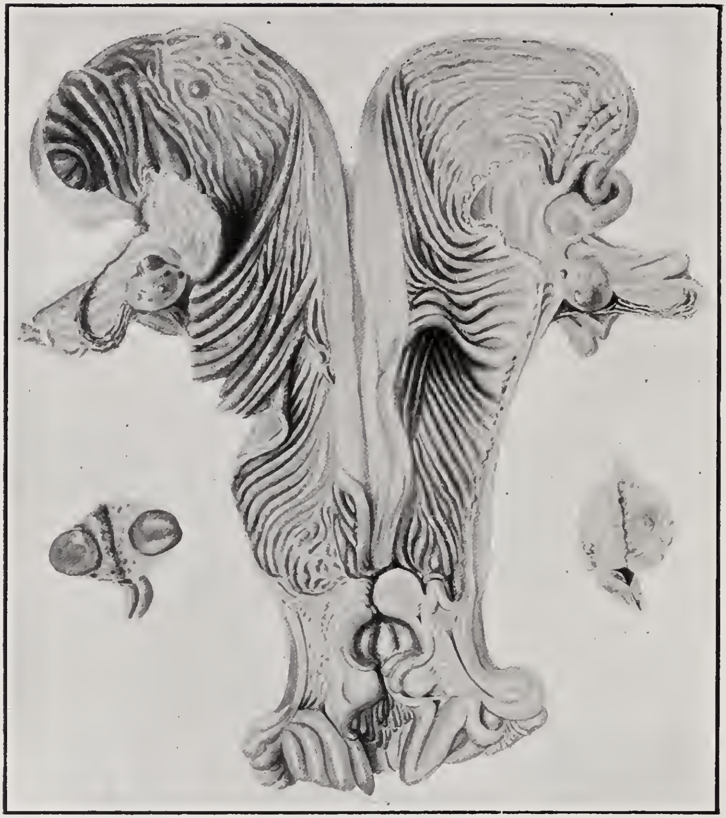


Fig. 11. (Case 11, App. I.)

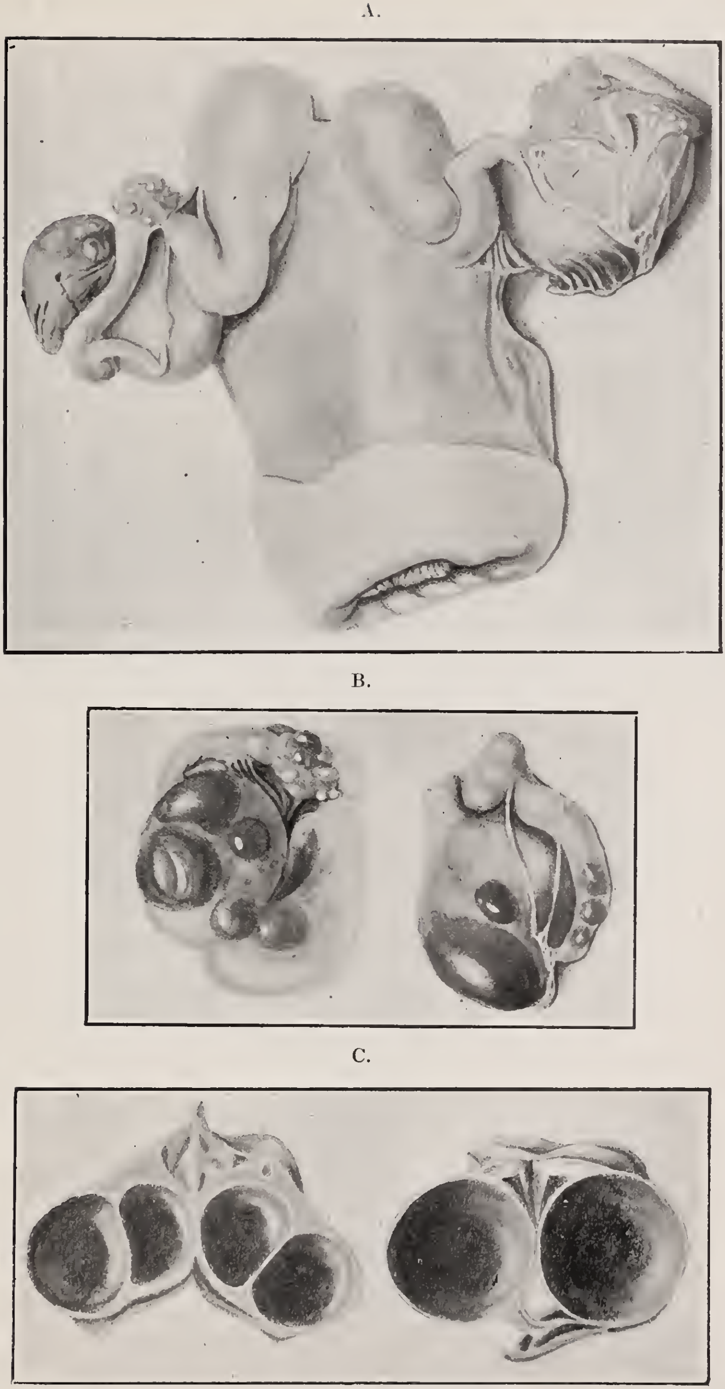


Fig. 12. (Case 12, App. I.)

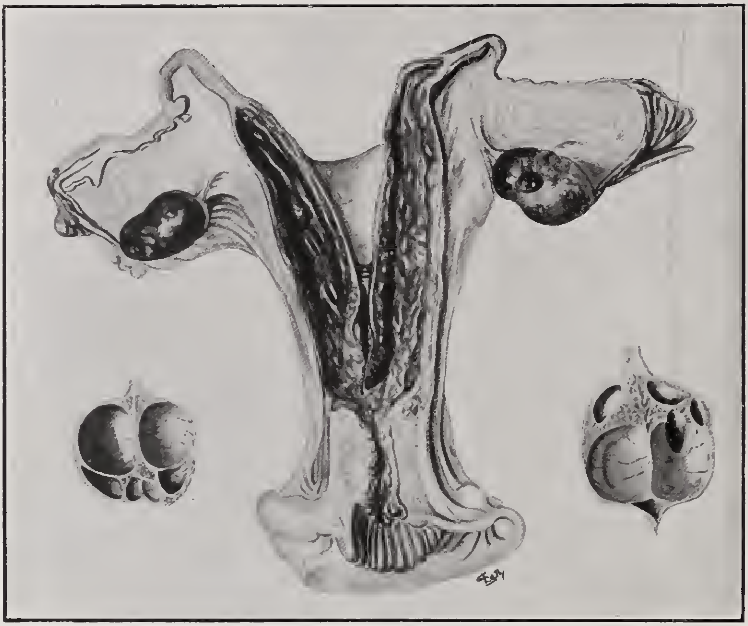


Fig. 13. (Case 13, App. I.)

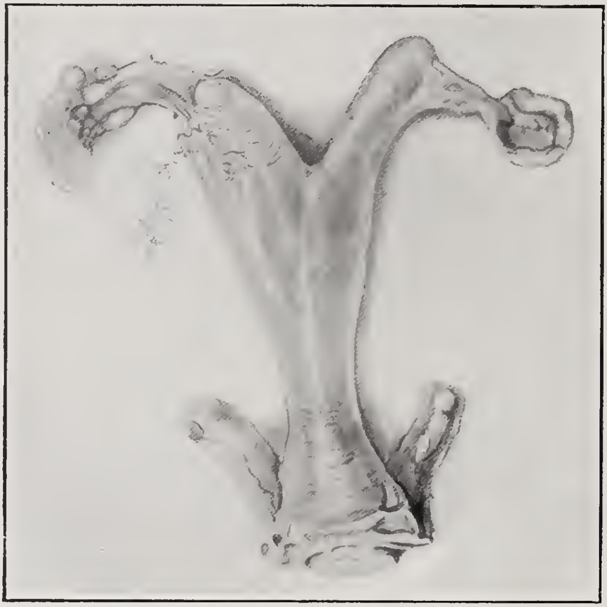


Fig. 14. (Case 14, App. I.)

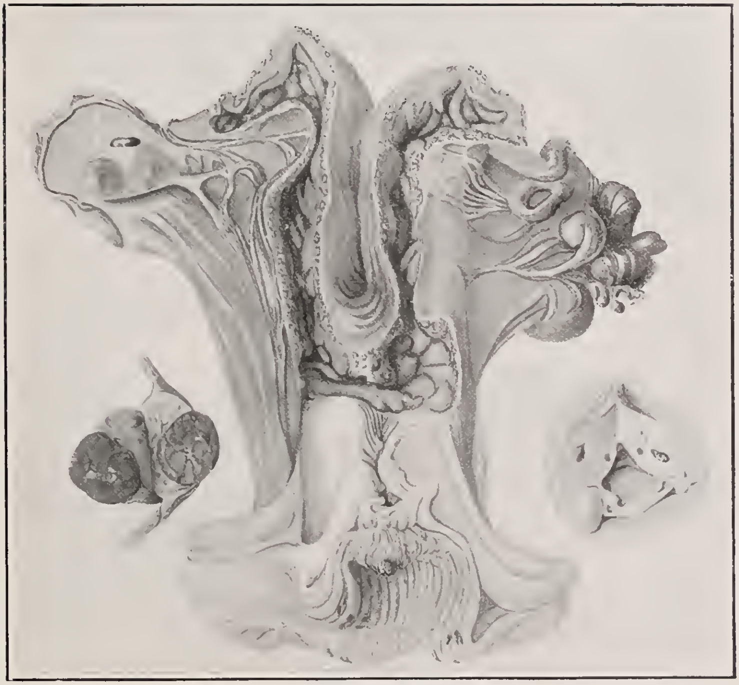
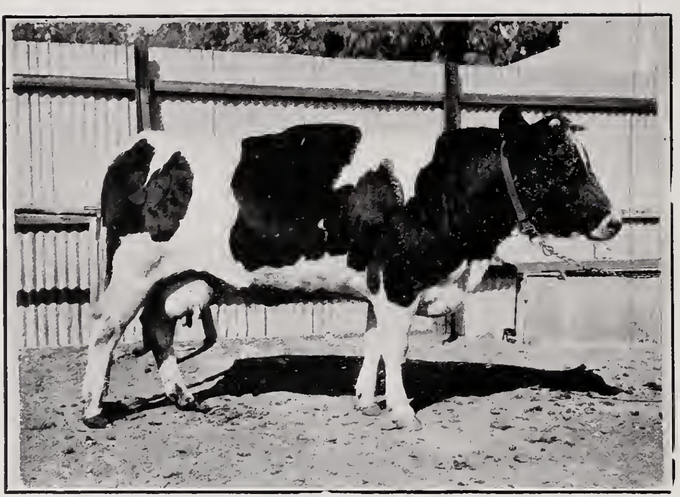


Fig. 15. (Case 15, App. I.)

A.



B.

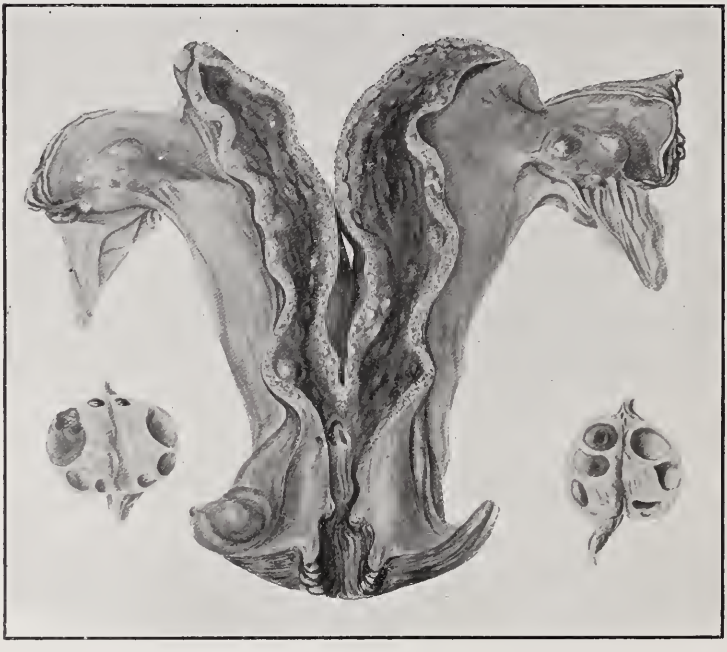


Fig. 16. (Case 16, App. I.)

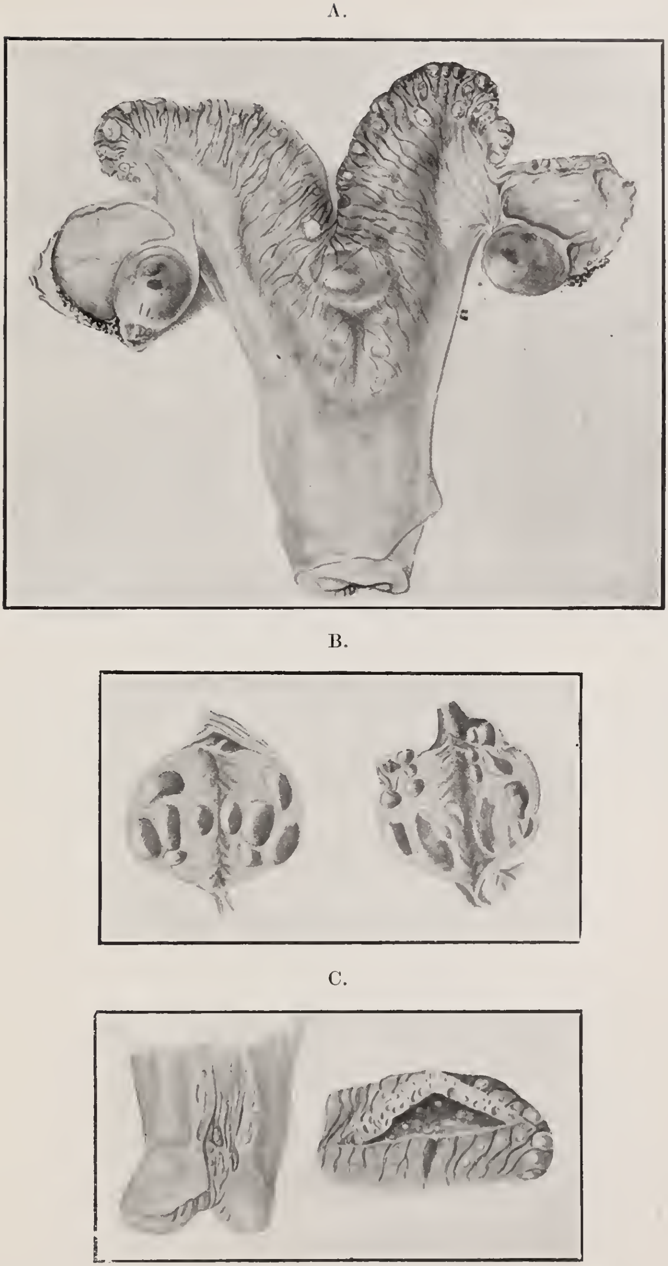
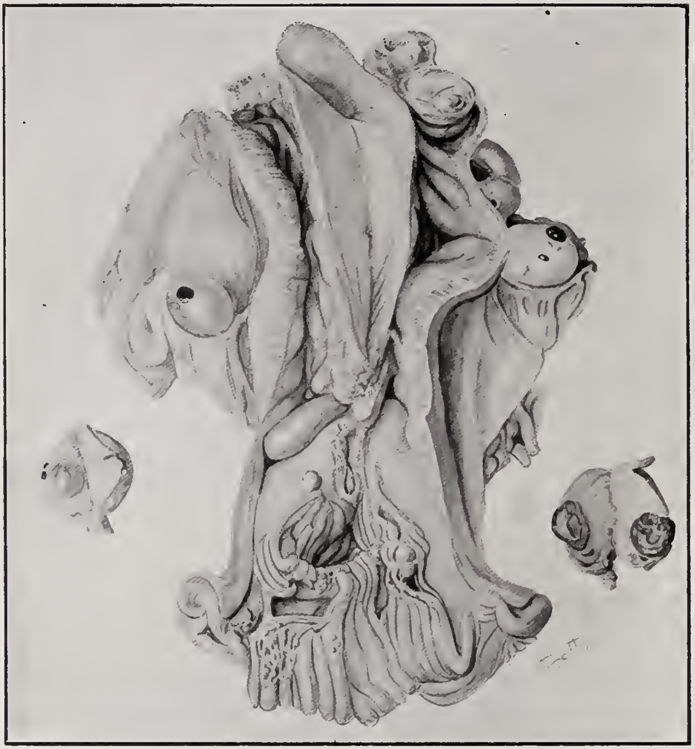


Fig. 17. (Case 17, App. I.)

A.



B.

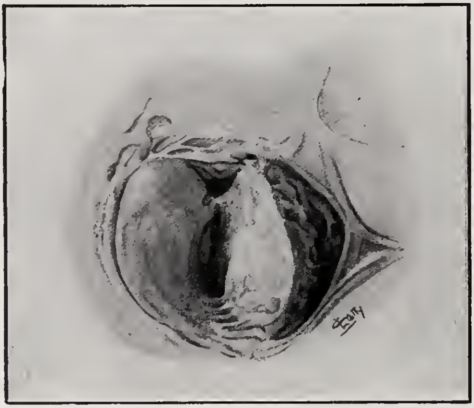


FIG. 18. (Case 18, App. I.)

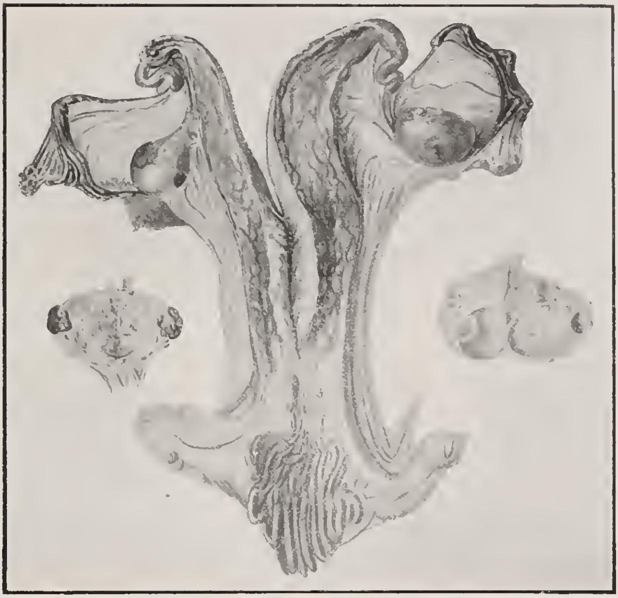


Fig. 19. (Case 19, App. I.)

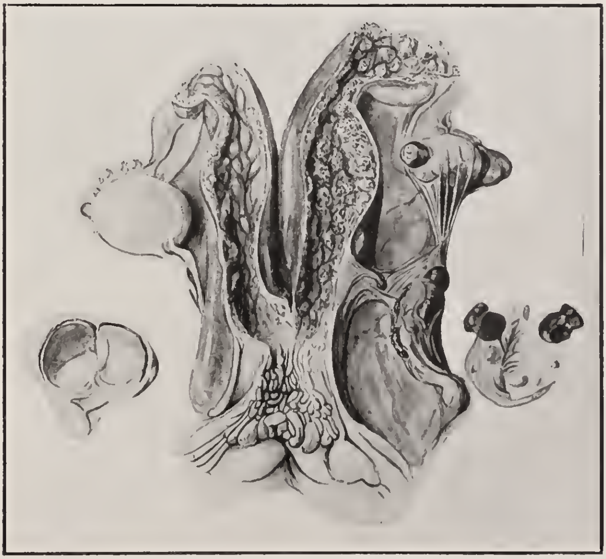


Fig. 20. (Case 20, App. I.)

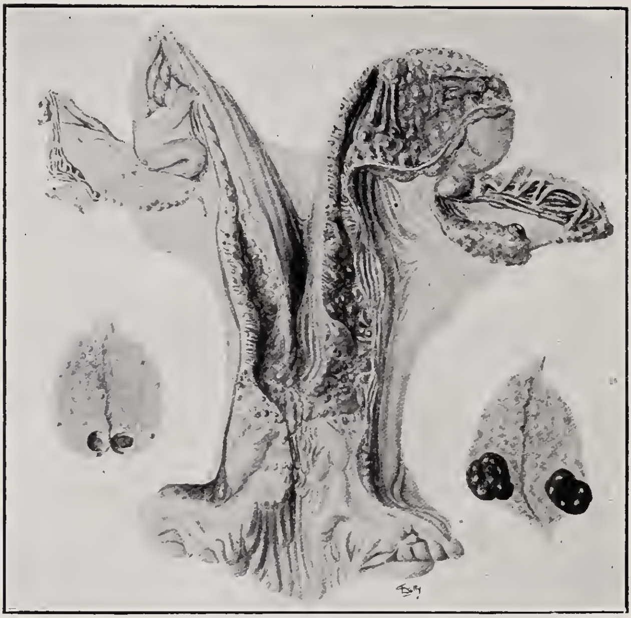


FIG. 21. (Case 21, App. I.)

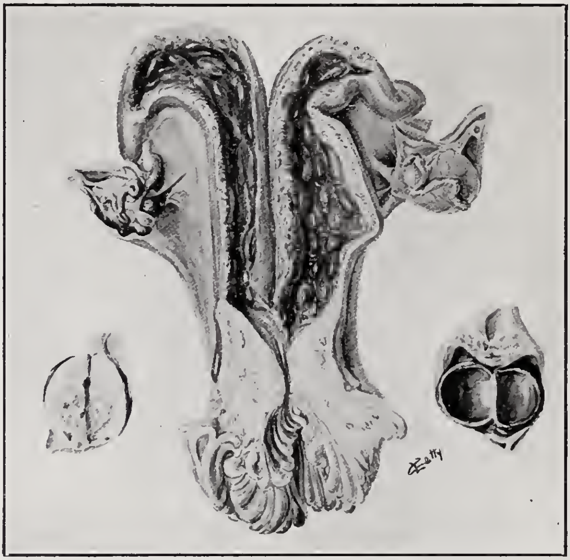


FIG. 22. (Case 22, App. I.)

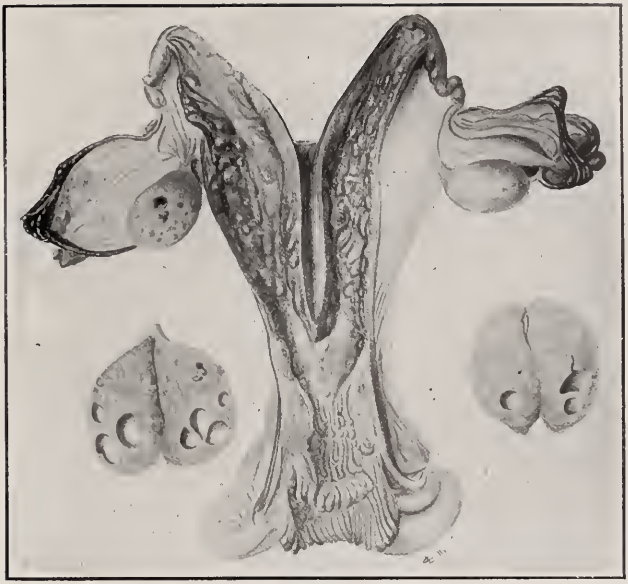


FIG. 23. (Case 23, App. I.)

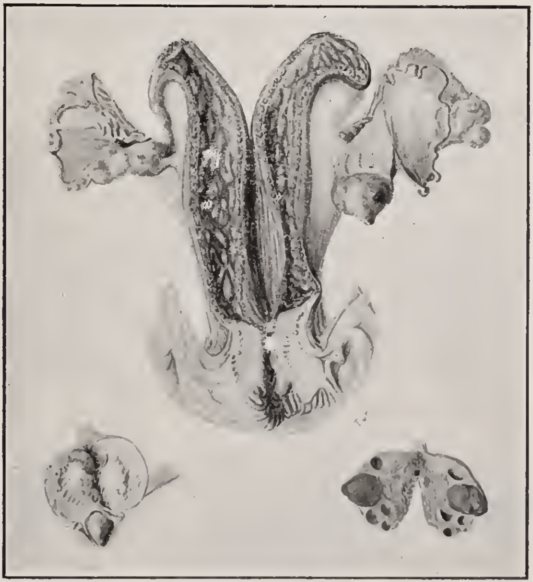


FIG. 24. (Case 24, App. I.)

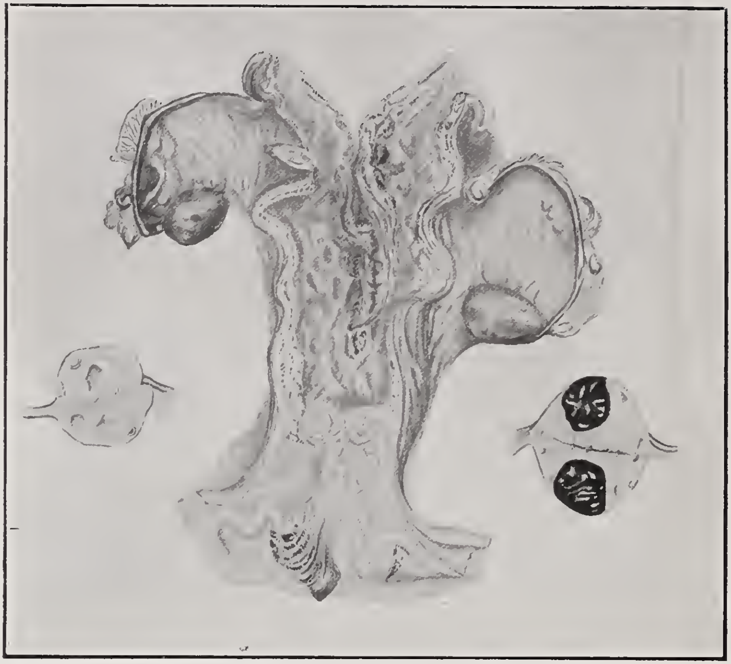


FIG. 25. (Case 25, App. I.)

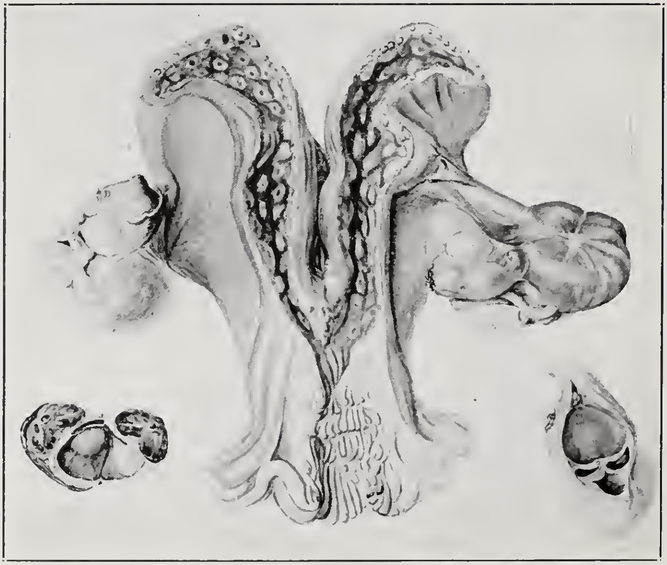


FIG. 26. (Case 29, App. I.)

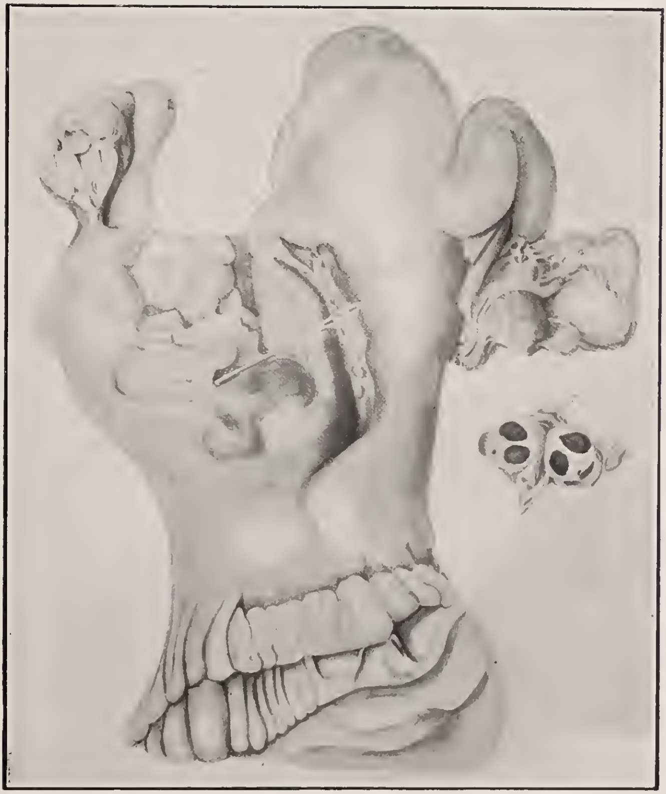
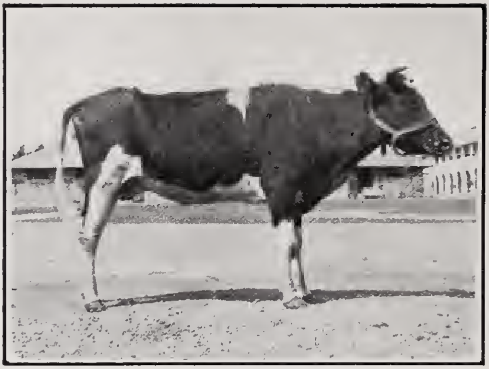


FIG. 27. (Case 26, App. I.)

A.



B.

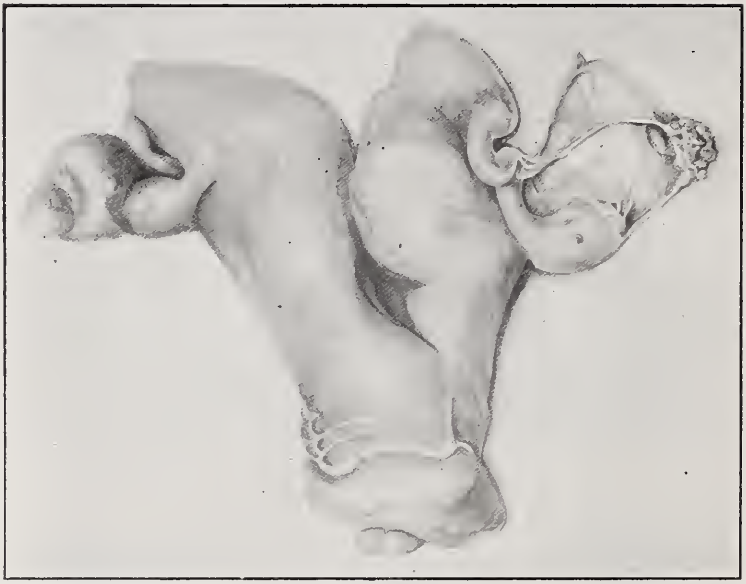


Fig. 28. (Case 27, App. I.)

C.

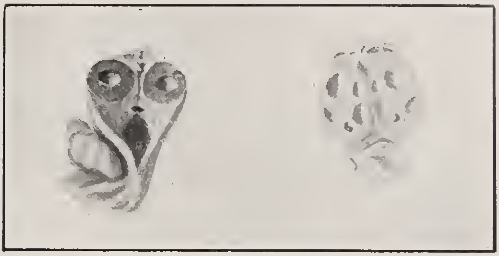


Fig. 28. (Case 27, App. I.)

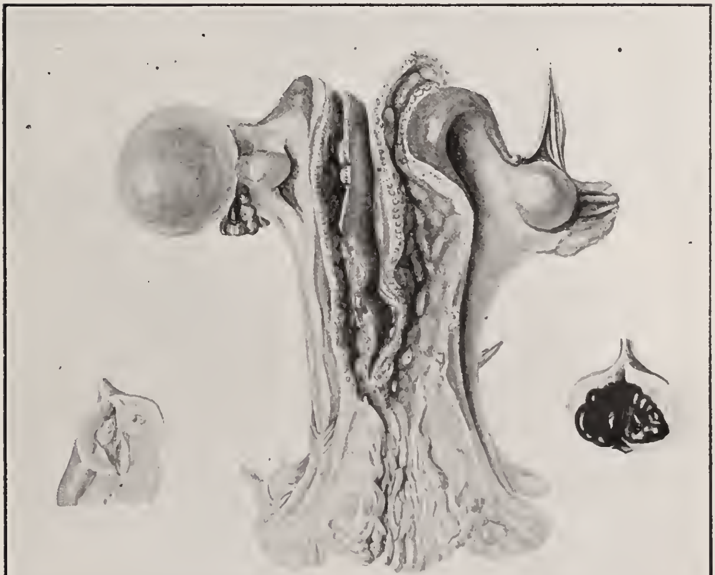
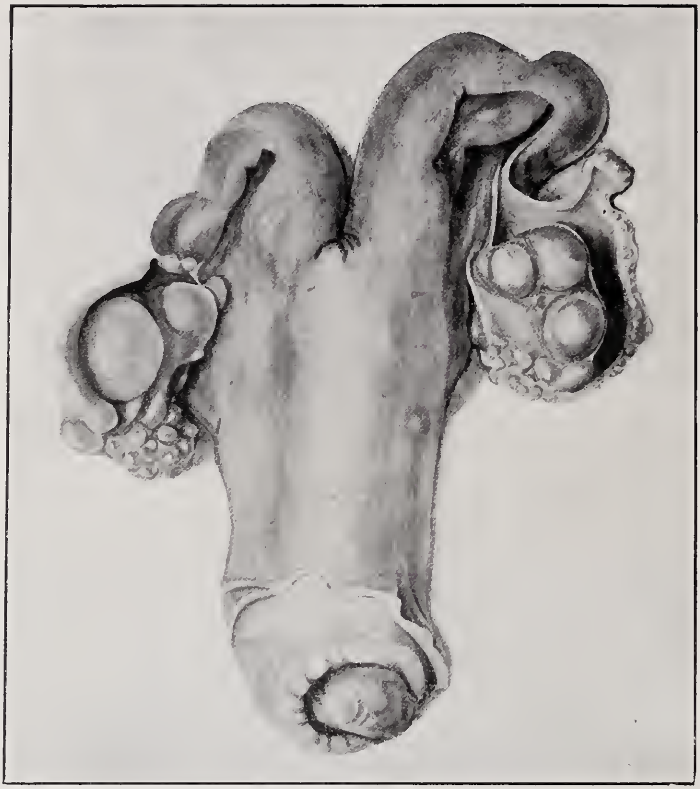


Fig. 29. (Case 28, App. I.)

A.



B.

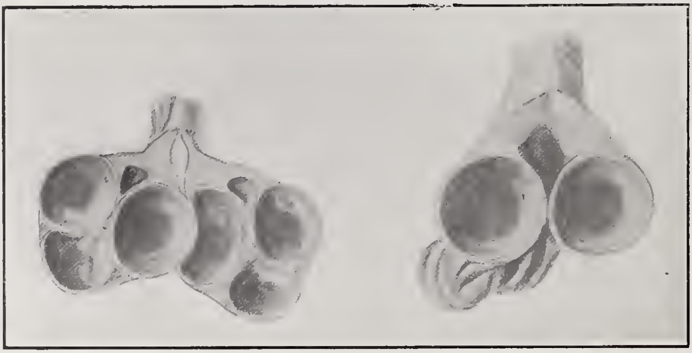


Fig. 30. (Case 31, App. 1.)

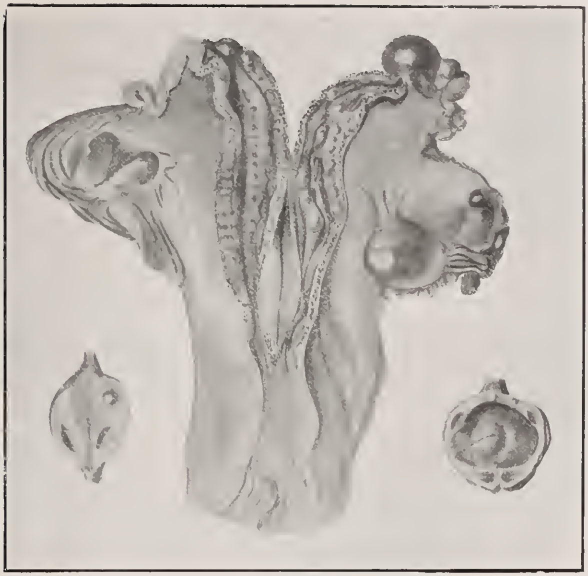


FIG. 31. (Case 32, App. I.)

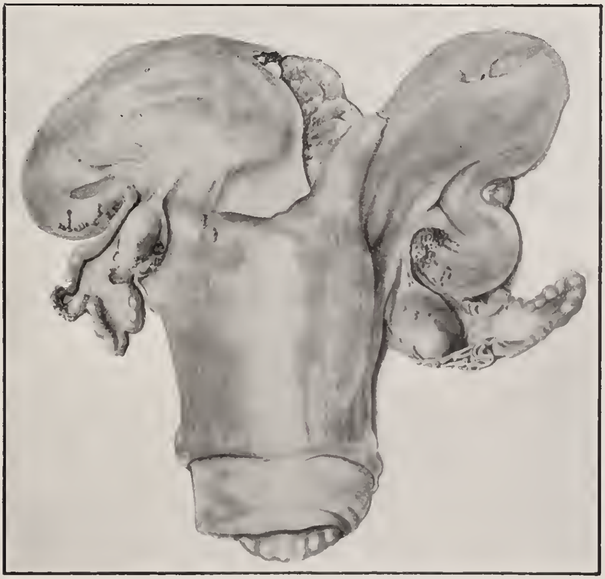


FIG. 32. (Case 33, App. I.)

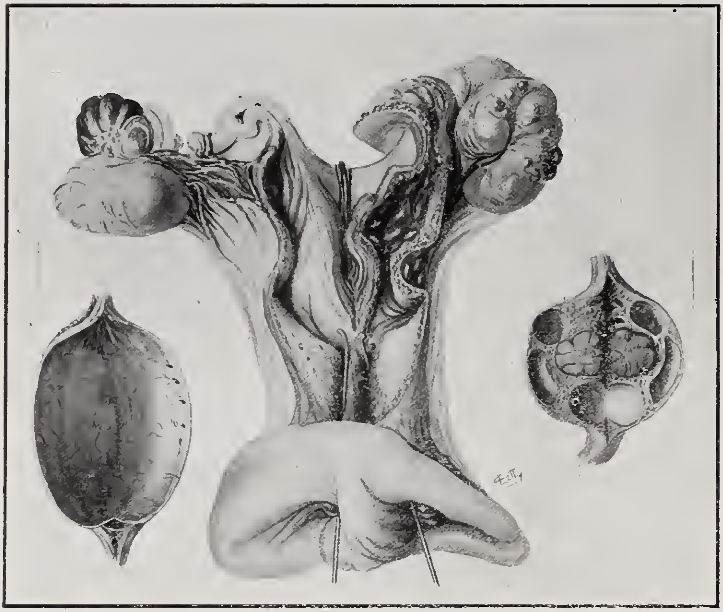


FIG. 33. (Case 34, App. I.)

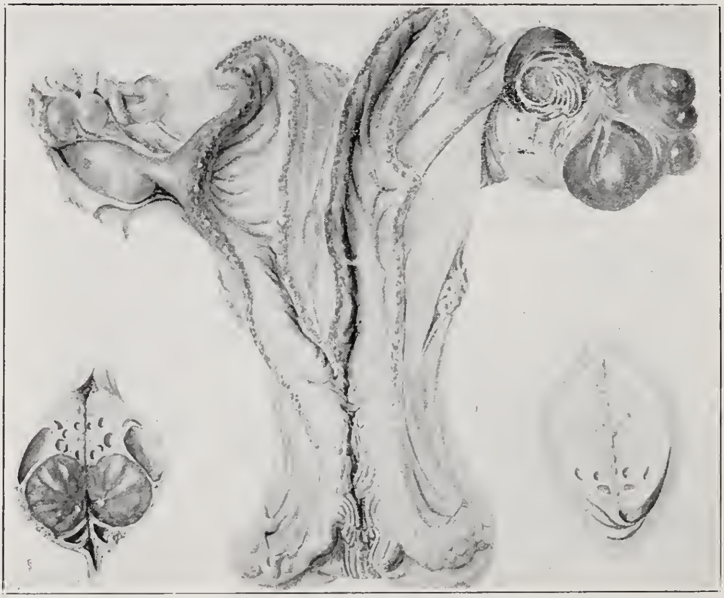


FIG. 34. (Case 35, App. I.)

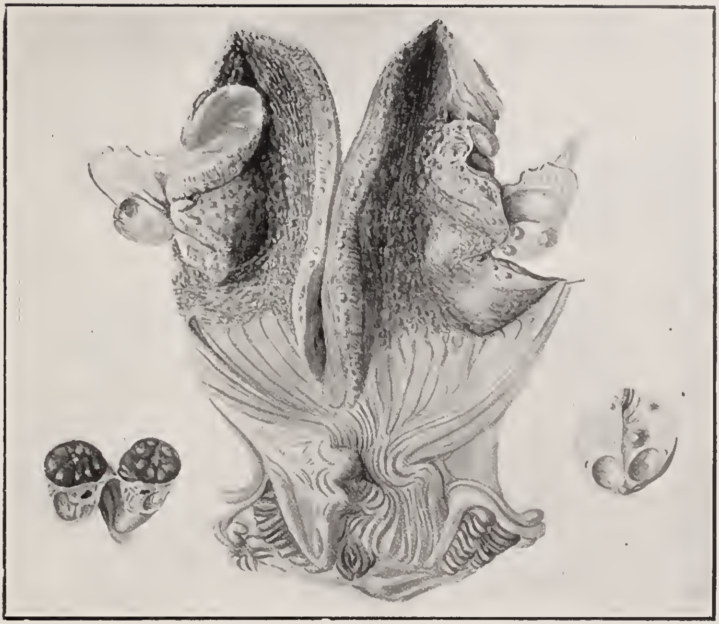


Fig. 35. (Case 36, App. I.)

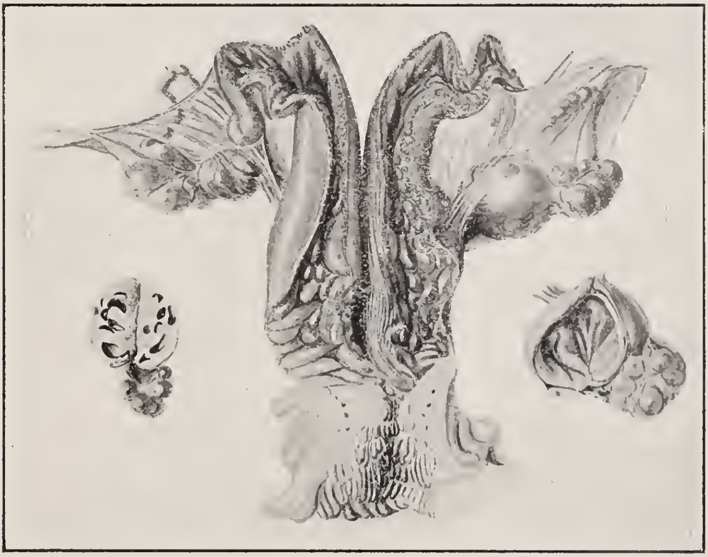


Fig. 36. (Case 37, App. I.)

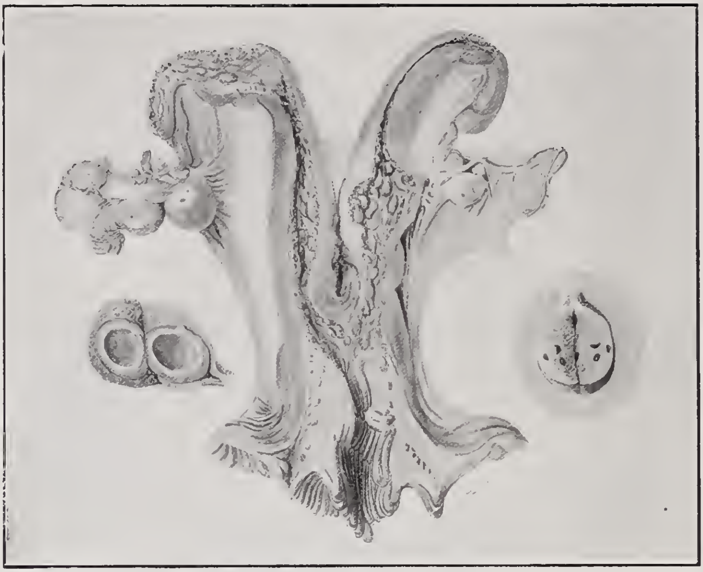


Fig. 37. (Case 38, App. I.)

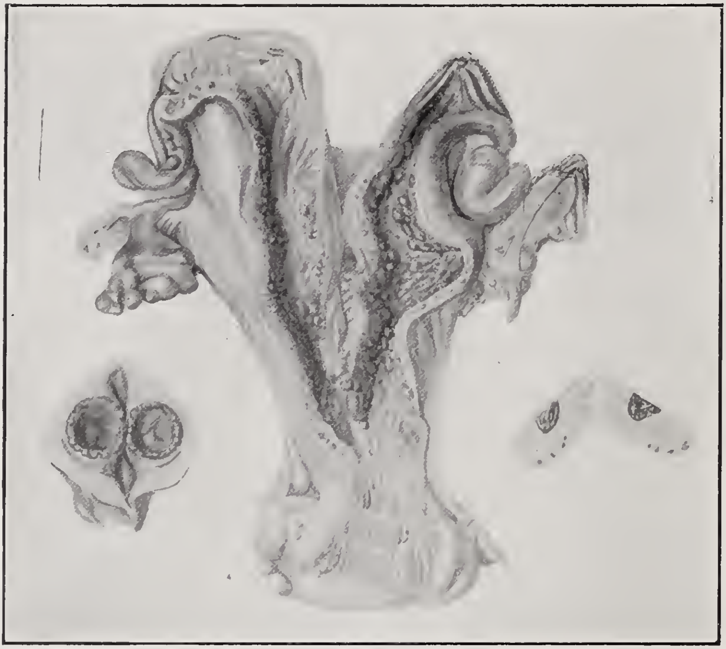


Fig. 38. (Case 39, App. I.)

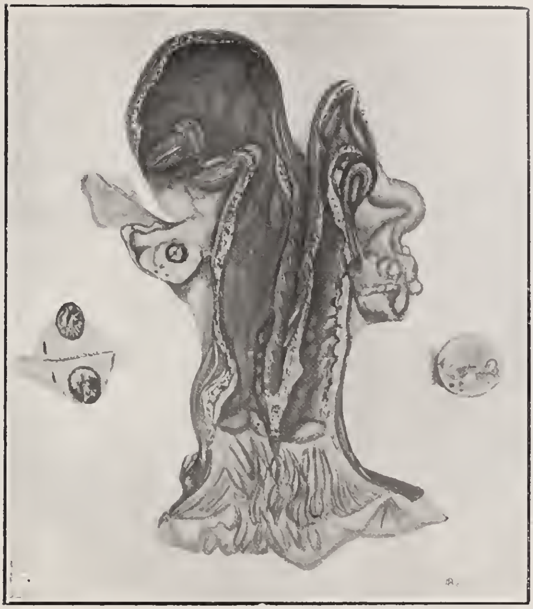


FIG. 39. (Case 40, App. I.)

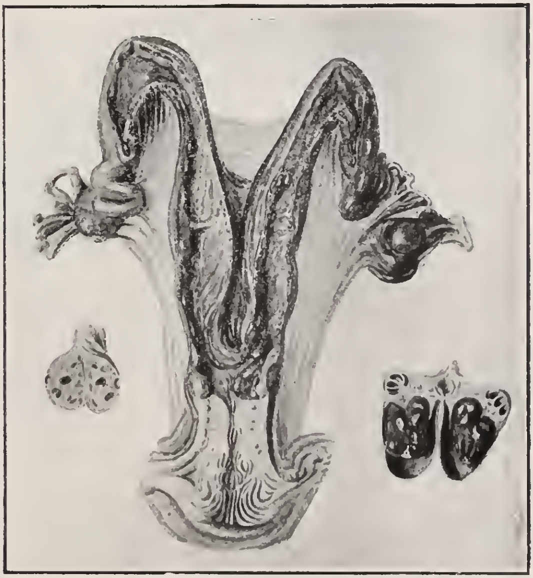


FIG. 40. (Case 41, App. I.)

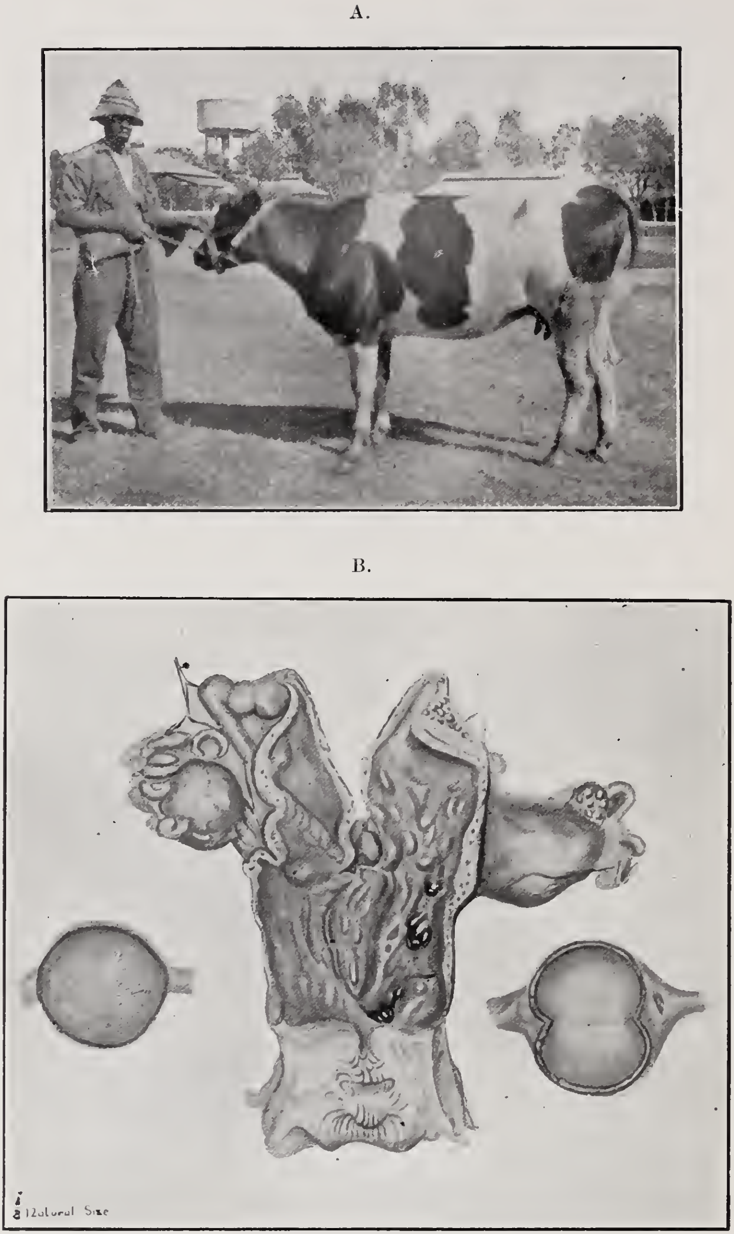


Fig. 41. (Case 42, App. I.)

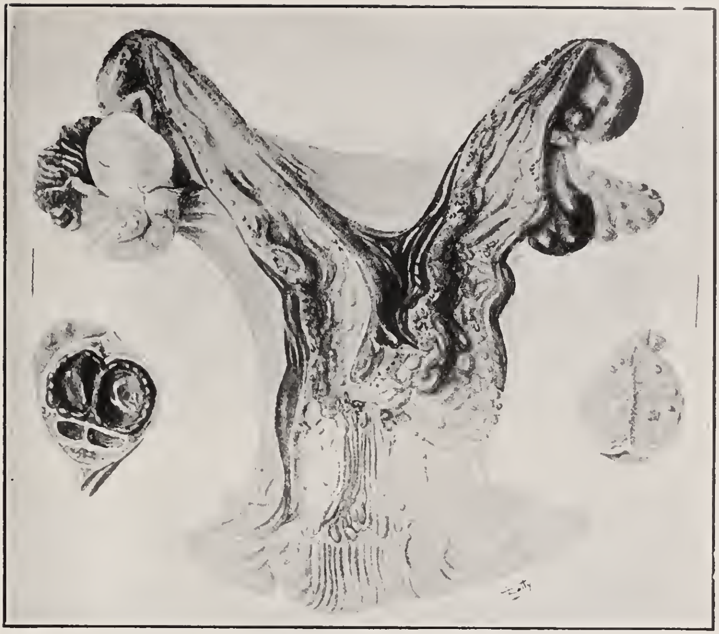


Fig. 42. (Case 43, App. I.)

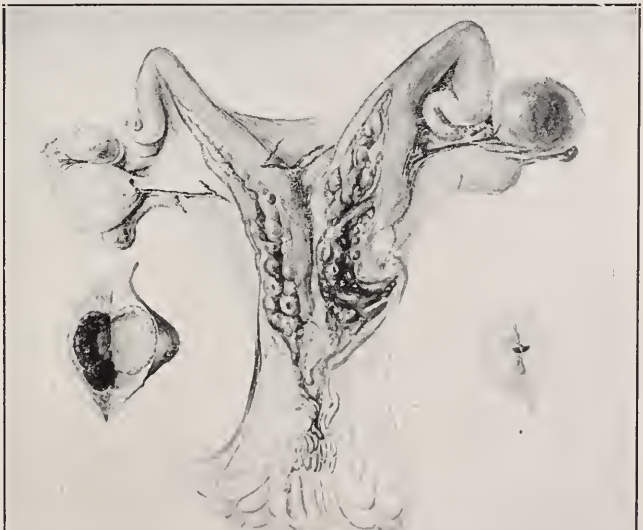
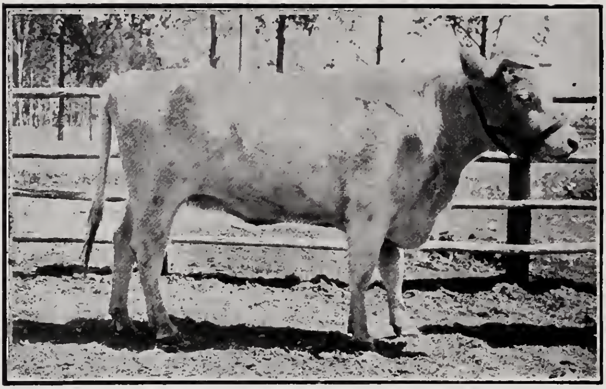


Fig. 43. (Case 44, App. I.)

A.



B.

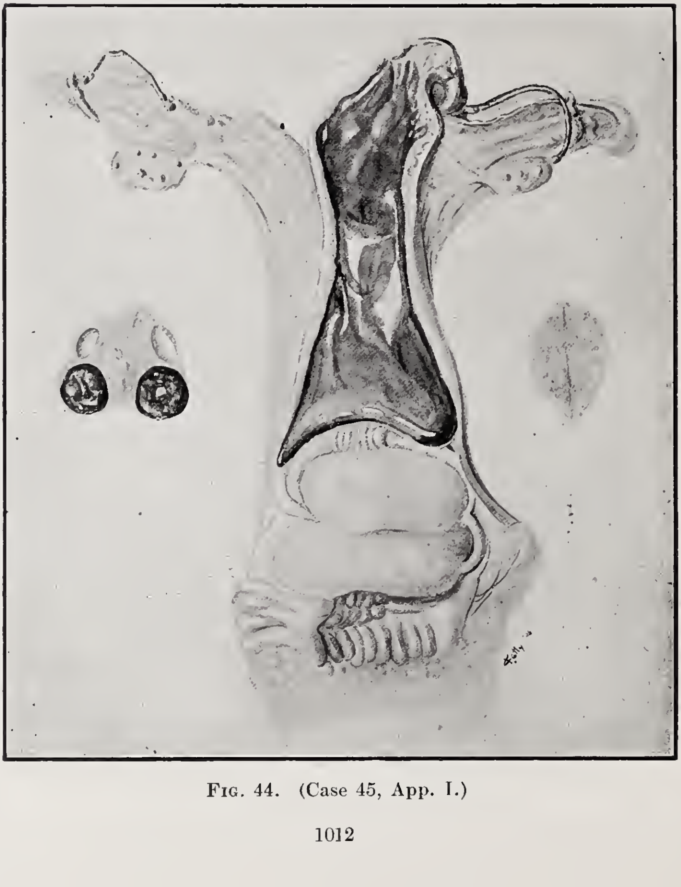


FIG. 44. (Case 45, App. I.)

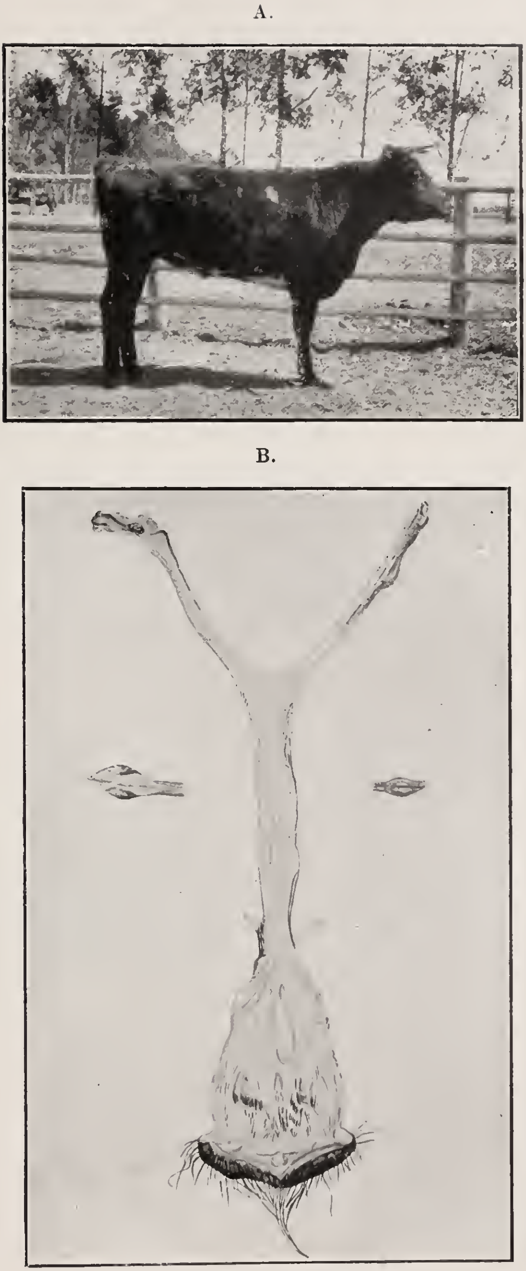


Fig. 45. (Case 46, App. I.)

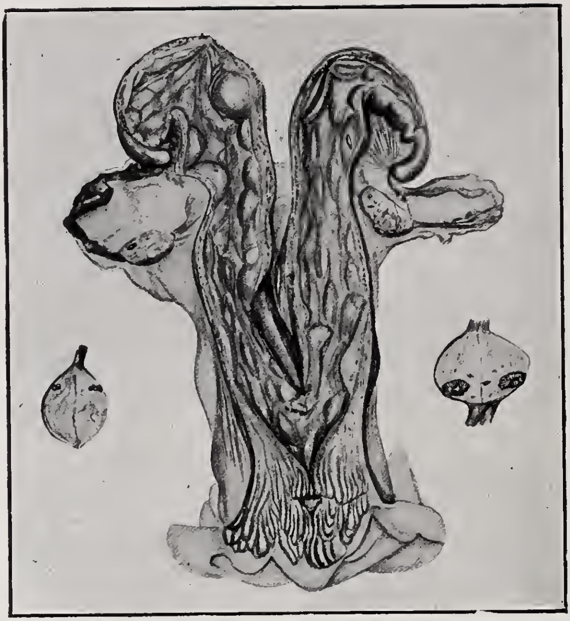


Fig. 46. (Case 47, App. I.)

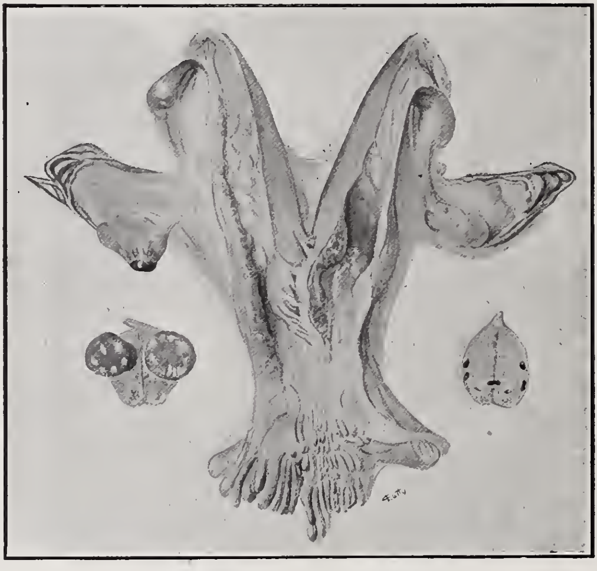


Fig. 47. (Case 48, App. I.)

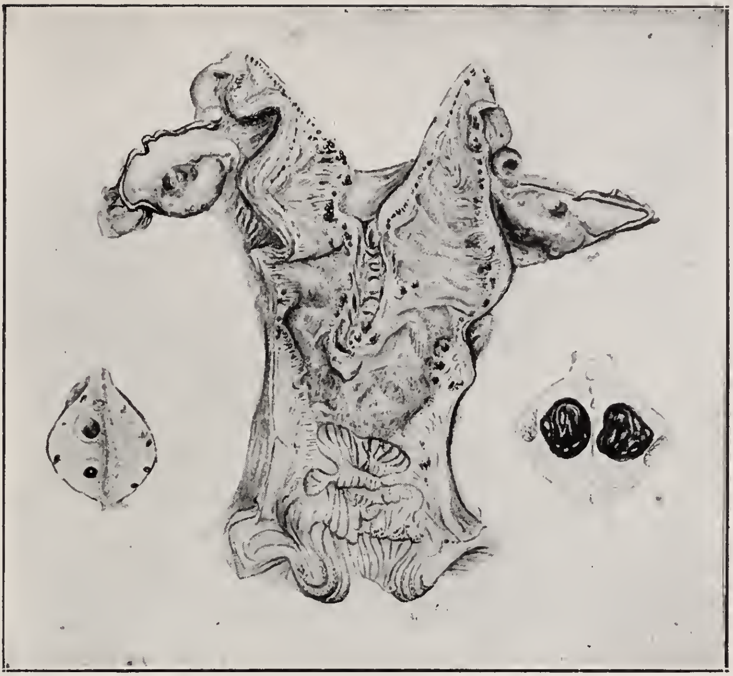


FIG. 48. (Case 49, App. I.)

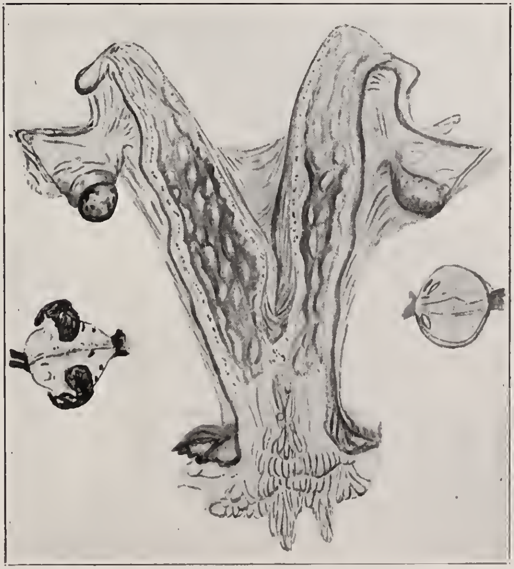


FIG. 49. (Case 50, App. I.)

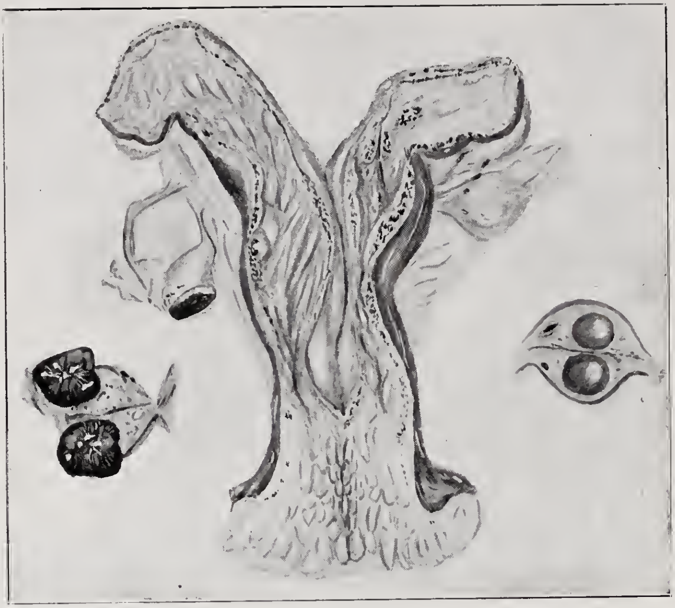


FIG. 50. (Case 51, App. I.)

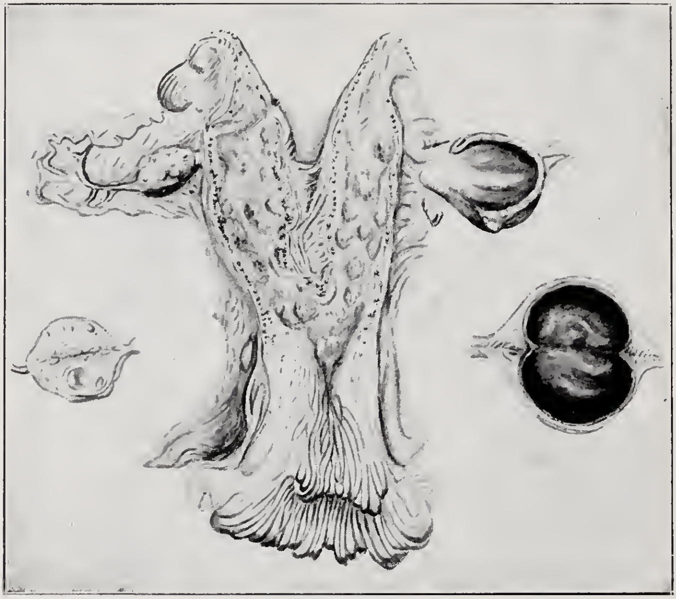


FIG. 51. (Case 52, App. I.)

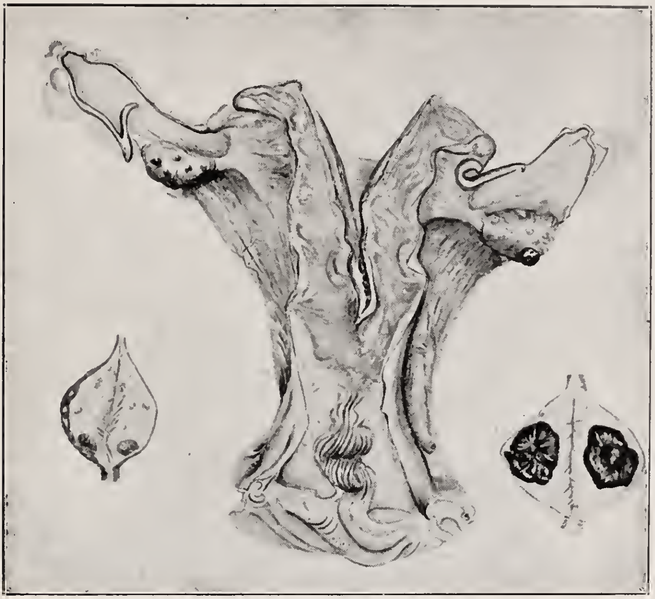


Fig. 52. (Case 53, App. I.)

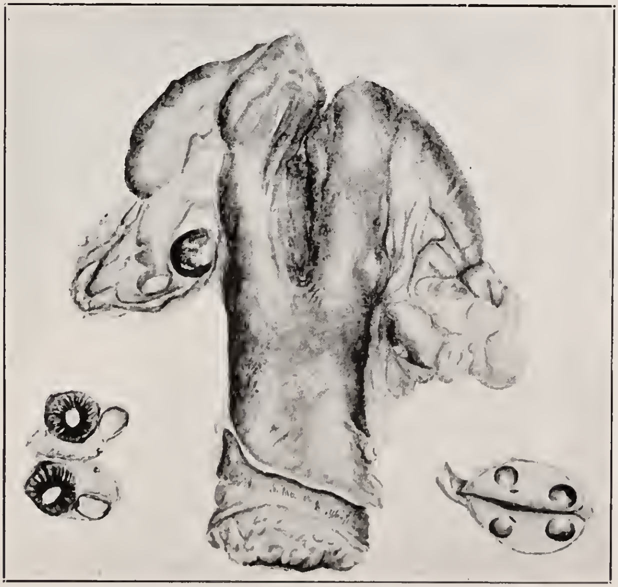


Fig. 53. (Case 54, App. I.)

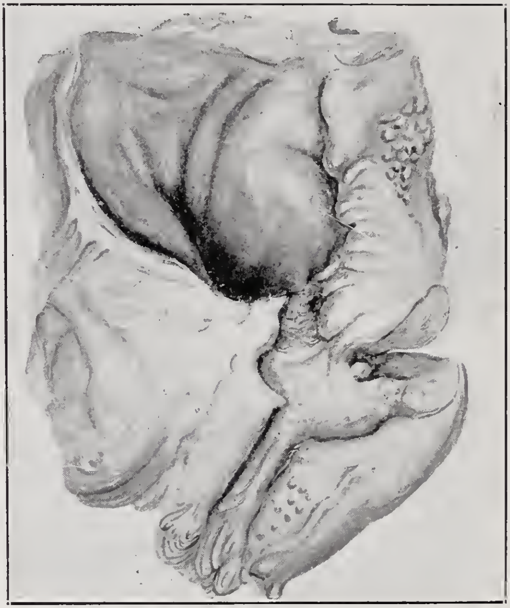


FIG. 54. (Case 55, App. I.)

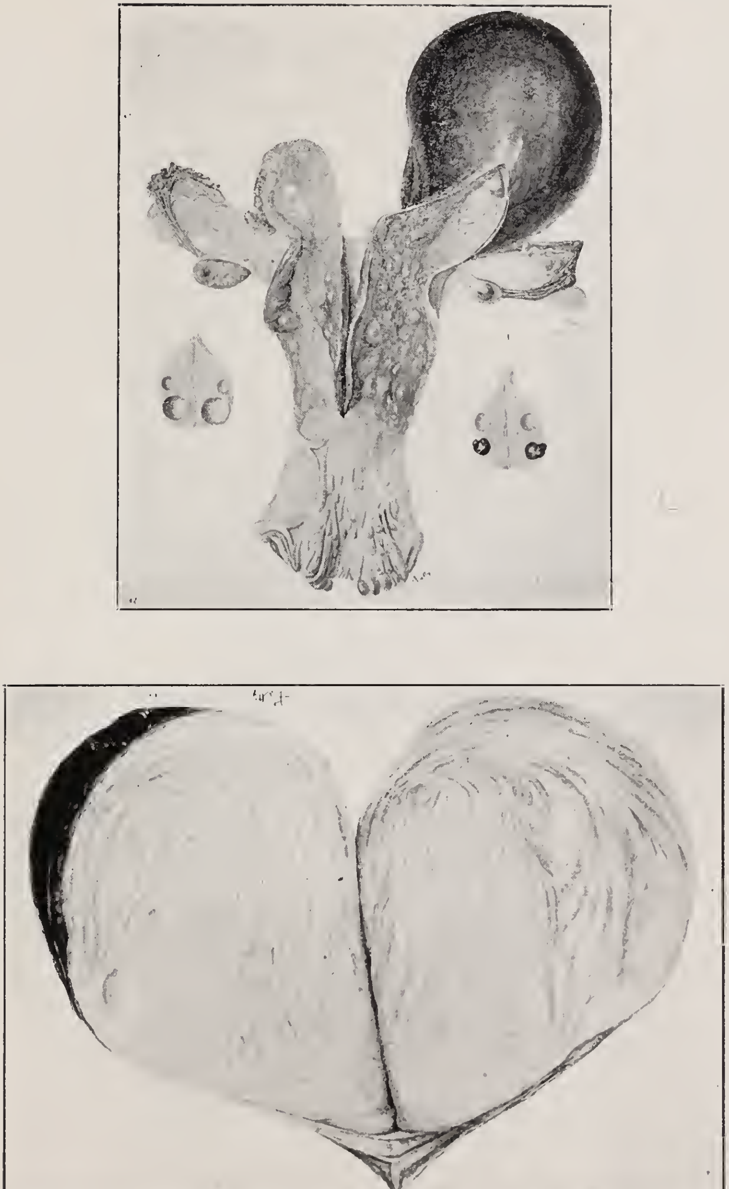


Fig. 55. (Case 56, App. I.)

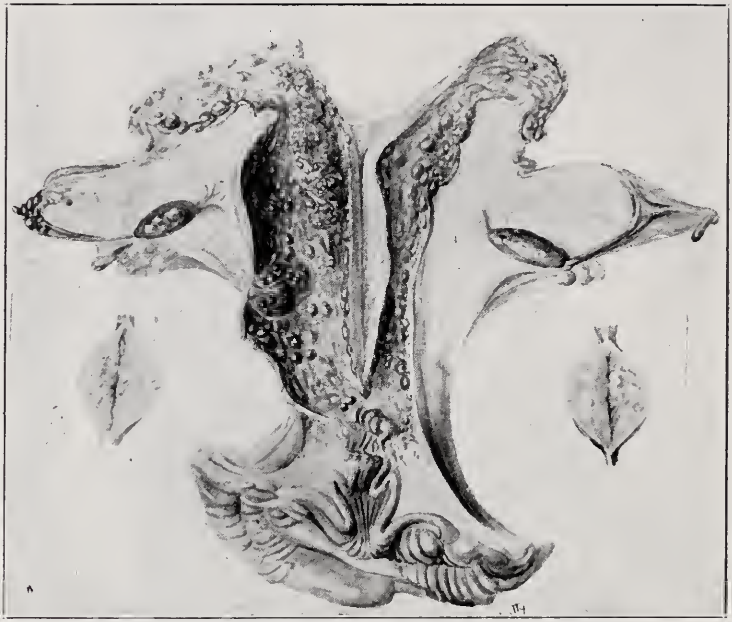


FIG. 56. (Case 57, App. I.)

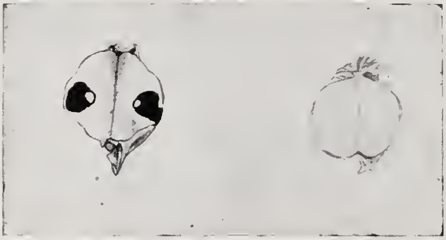


FIG. 57. (Case 58, App. I.)

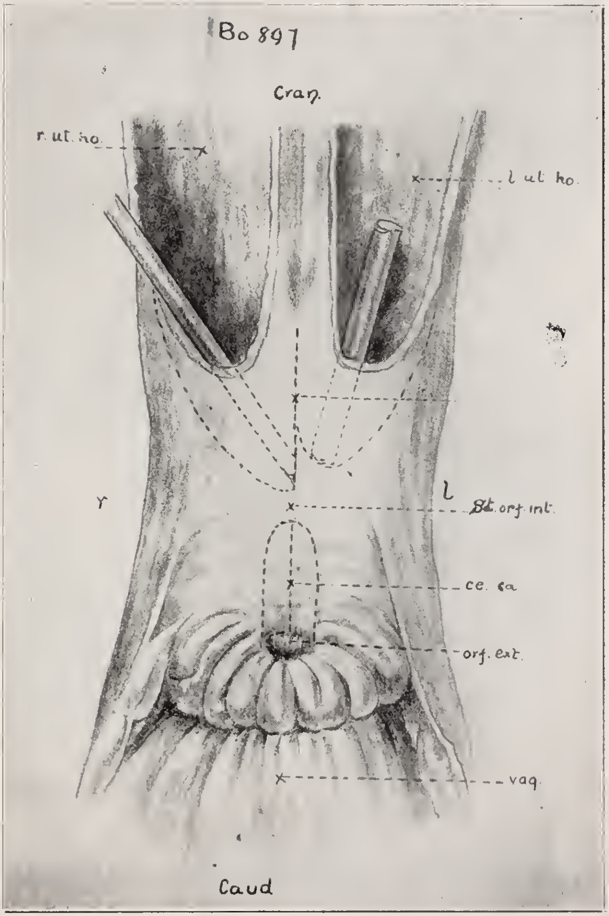


Fig. 58. (Case 59, App. I.)

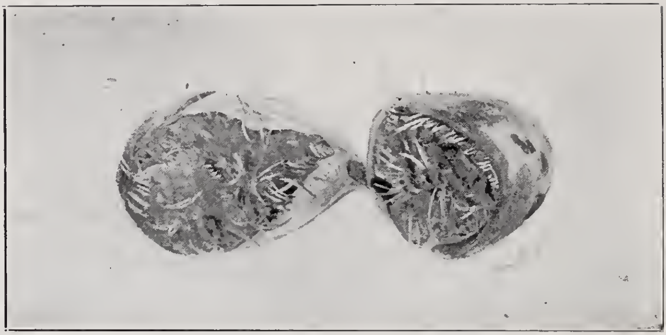


Fig. 59. (Case 60, App. I.)

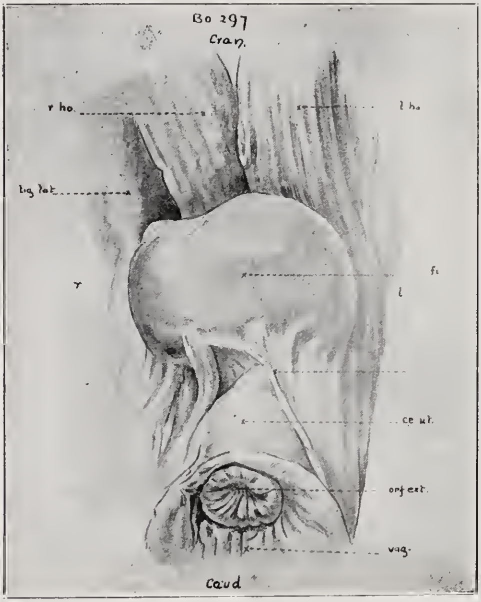


Fig. 60. (Case 61, App. I.)

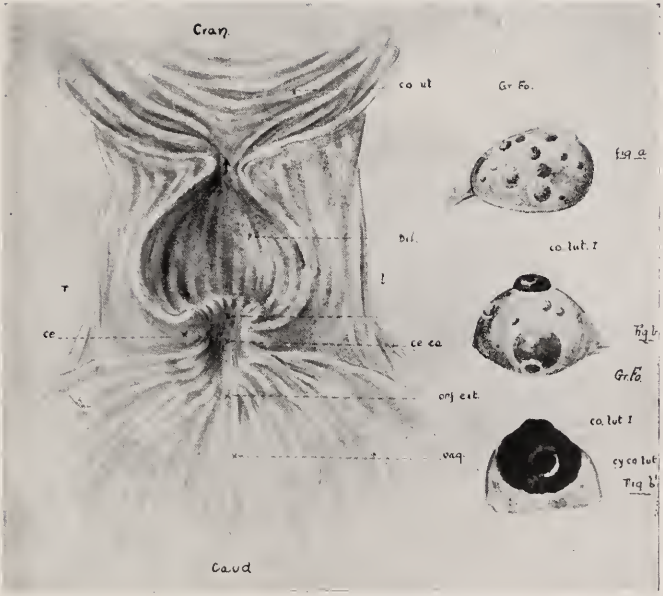


FIG. 61. (Case 62, App. I.)

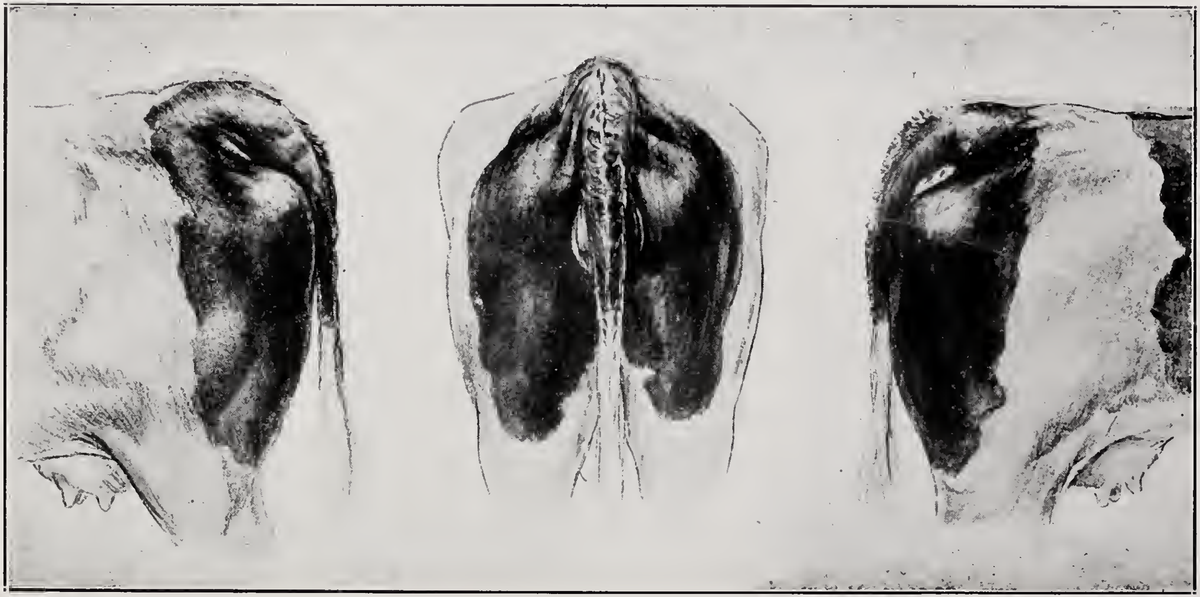


Fig. c. Fig. a. Fig. b.

Fig. 62. (Case 63, App. I.)

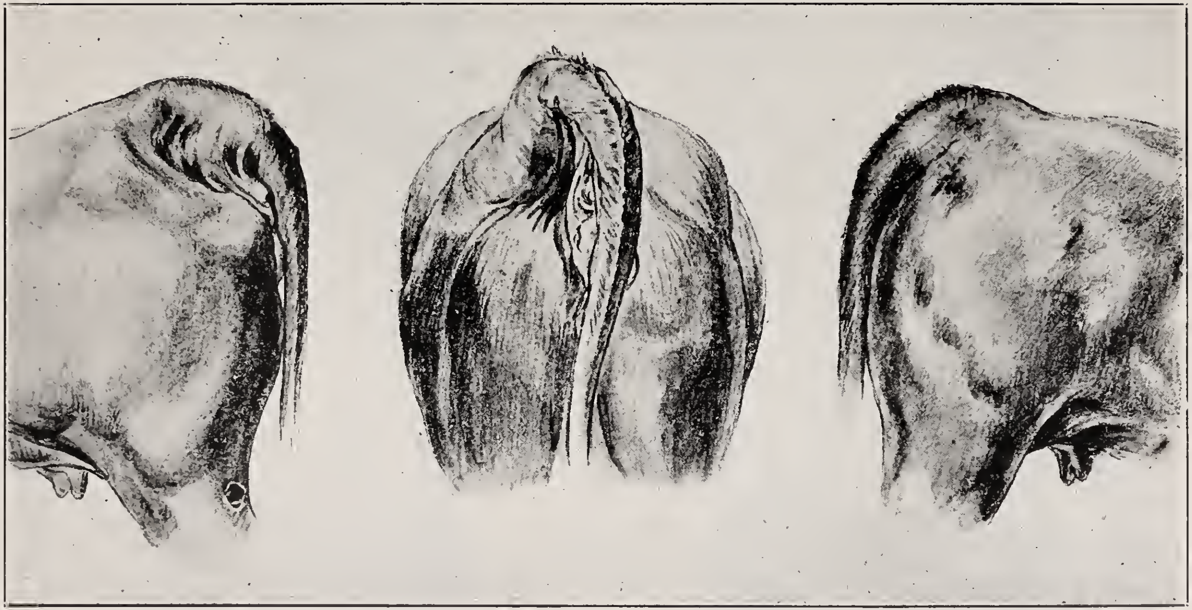


Fig. c. Fig. a. Fig. b.

FIG. 63. (Case 64, App. I.)

APPENDIX III.

In this appendix are shown microphotographs which represent the pathological changes in the genitalia described in the text.

The photographs were taken by Mr. Theo. Meyer, of this Institution.

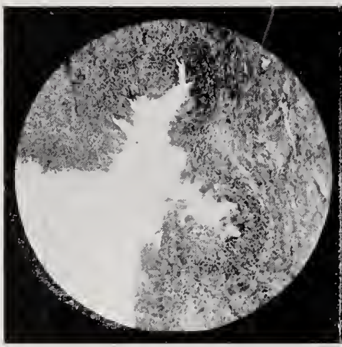


Fig. 1.

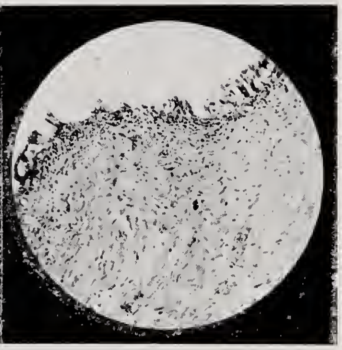


Fig. 2.

Fig. 1.—Section of the cervix, showing epithelial desquamation and round-cell infiltration (acute stage). × 66.

Fig. 2.—Section of the cervix, showing epithelial desquamation and some round-cell infiltration. Organization of the cellular elements has taken place (sclerosis). × 78.

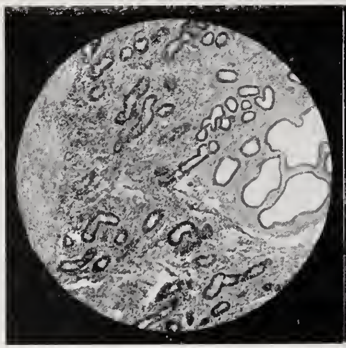


Fig. 3.

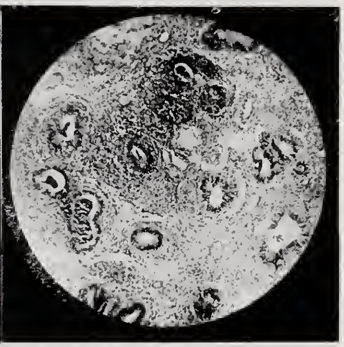


Fig. 4.

Fig. 3.—Section of the mucosa of the uterus, showing periglandular cellular infiltration; some of the glands are filled with cells and cell débris, while others are normal. × 66.

Fig. 4.—Section of the mucosa of the uterus, showing some enlarged glands, the commencement of metritis chronica catarrhalis cystica. × 66.

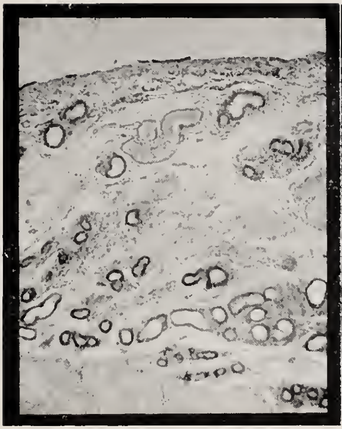


FIG. 5.

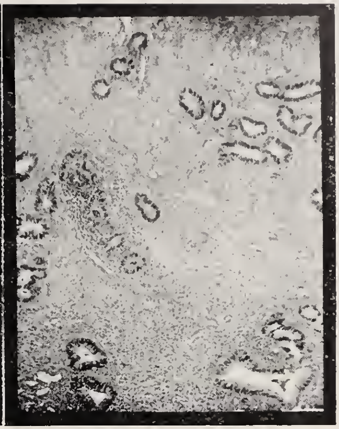


FIG. 6.

FIG. 5.—Section of the mucosa of the uterus, showing oedema, vascular dilatation, and commencing cyst formation in some of the deeper glands. × 70.

FIG. 6.—Section of the mucosa of the uterus, showing periglandular cellular infiltration; some of the glands are filled with round cells and cell débris, while others are normal. × 70.

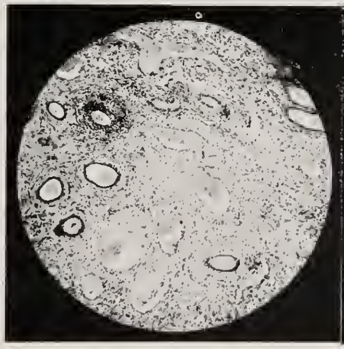


FIG. 7.

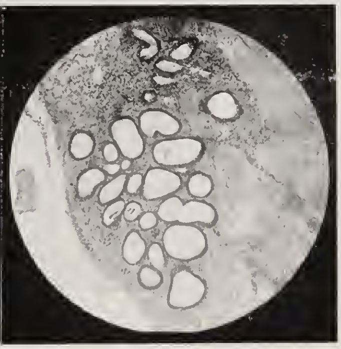


FIG. 8.

FIG. 7.—Section of the mucosa of the uterus, showing periglandular round-cell infiltration and vascular dilatation. × 66.

FIG. 8.—Section through the zona muscularis of the uterus just underneath the mucosa, showing a group of enlarged uterine glands mostly with flattened epithelium. × 66.

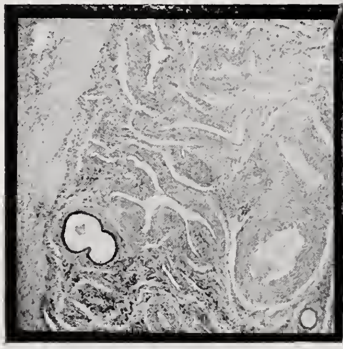


FIG. 9.

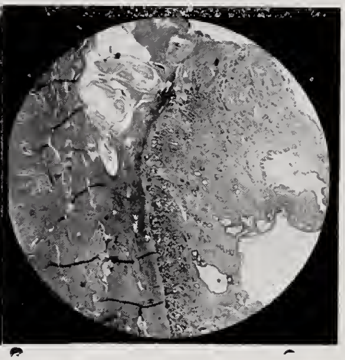


FIG. 10.

FIG. 9.—Section through the zona muscularis of the uterus just underneath the mucosa, showing a single enlarged uterine gland. × 66.

FIG. 10.—Section through the uterine wall, showing several cysts. Many of the glands show enlargement, while others are normal. × 10.

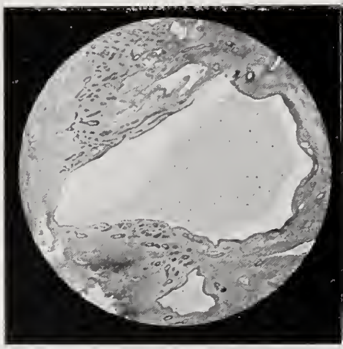


FIG. 11.

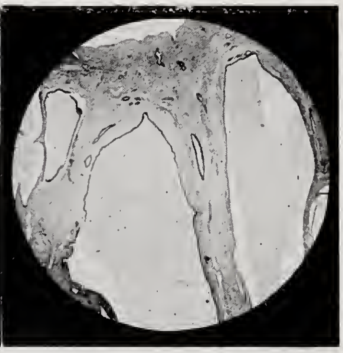
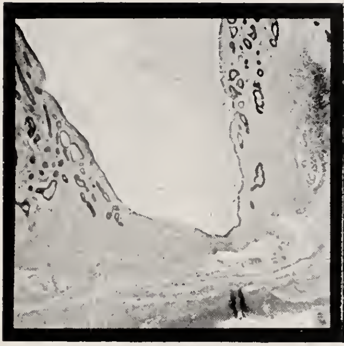


FIG. 12.

FIG. 11.—Section through portion of the uterine mucosa, showing large cyst. lined with epithelium. × 20.

FIG. 12.—Section through a portion of the uterine wall, showing a large cyst encroaching on the zona muscularis. There are smaller cysts on the right and left. × 15.



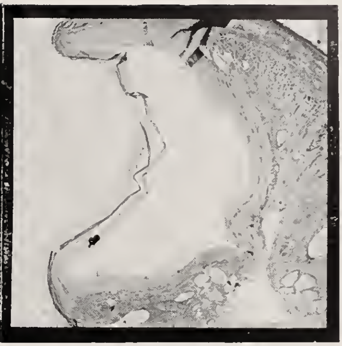
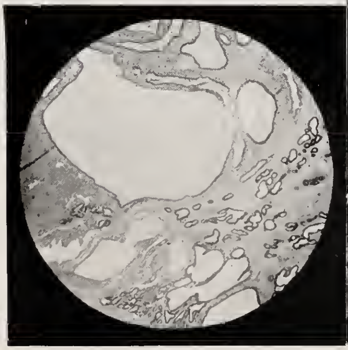


FIG. 13.

FIG. 14.

FIG. 13.—Portion of section shown in Fig. 11. × 50.

FIG. 14.—Section through the uterine wall, showing several small cysts, and a large cyst the capsule of which is collapsed owing to its contents being evacuated. × 15.



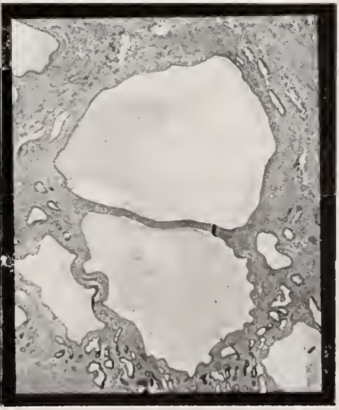


FIG. 15.

FIG. 16.

FIGS. 15 and 16.—Section through mucosa of the uterus, showing cystic change. 15, × 20; 16, × 38.

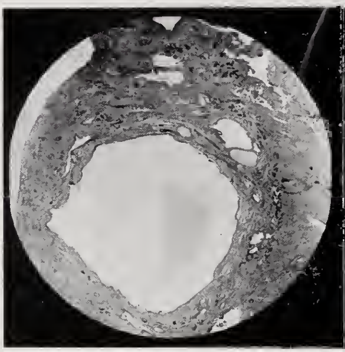


FIG. 17.

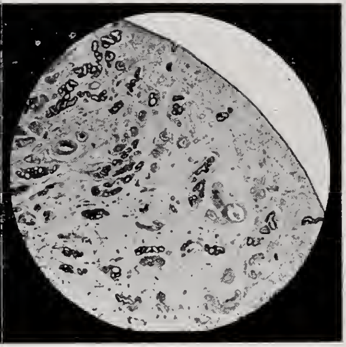


FIG. 18.

FIG. 17.—Section through mucosa of the uterus, showing cystic change. × 12.

FIG. 18.—Section through the mucosa of the uterus, showing oedema and group isolation of the uterine glands. The epithelium shows local stratification. × 20.

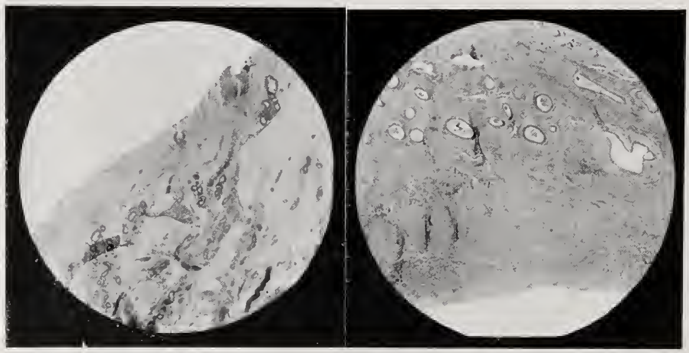


FIG. 19.

FIG. 20.

FIGS. 19 and 20.—Section through the mucosa of a pyometric uterus, showing absence of epithelium, cellular infiltration, and reduction in the number of uterine glands. × 20.

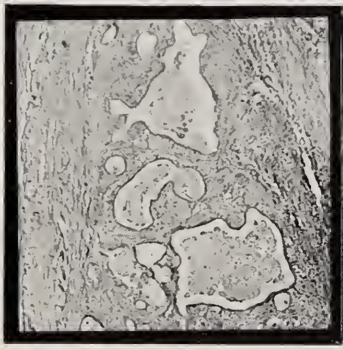


FIG. 21.

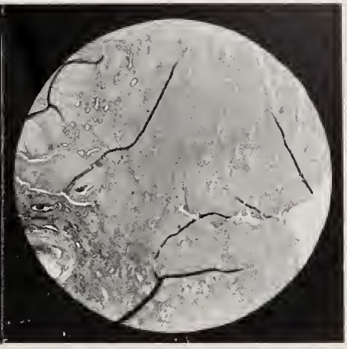


FIG. 22.

FIG. 21.—Section through the mucosa of a pyometric uterus, showing the glands towards the surface filled with purulent débris, with the epithelium desquamated. The epithelium lining the uterus has disappeared. There are some normal glands towards the zona muscularis. × 20.

FIG. 22.—Section through pyometric uterine wall at the junction of the mucosa and zona muscularis, showing enlarged uterine glands filled with desquamated epithelial cells and débris. × 75.

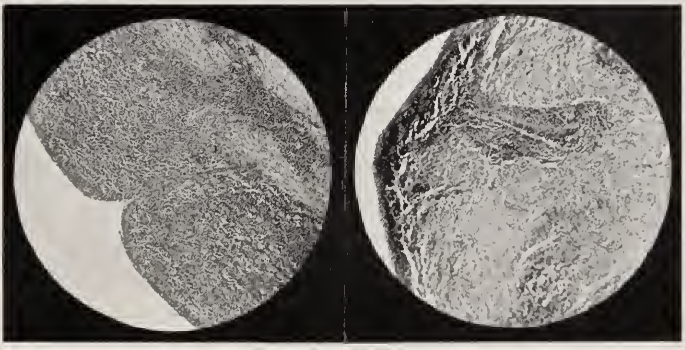


FIG. 23. FIG. 24.

FIGS. 23 and 24.—Section through the mucosa of a pyometric uterus, showing absence of glands and transformation of the normal mucosa to granulation tissue. × 66.

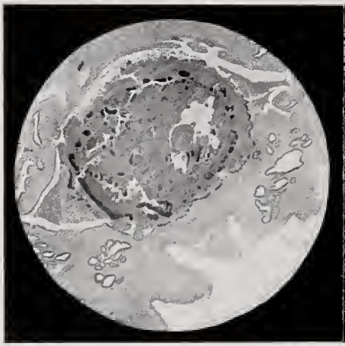


Fig. 25.

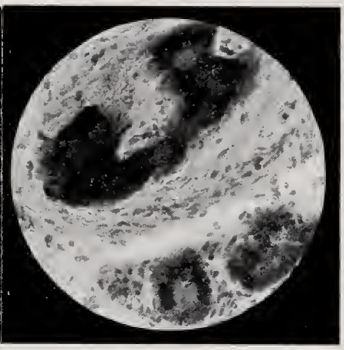


Fig. 26.

Fig. 25.—Section through the mucosa of the uterus, showing an enlarged gland filled with inspissated débris which shows commencing calcification. Towards the right there is a mass of closely packed neutrophiles. × 20.

Fig. 26.—Section through the mucosa of the uterus, showing calcification of the wall of two capillaries. × 140.

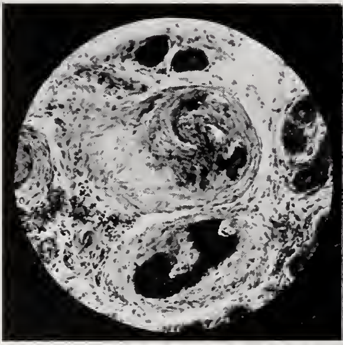


Fig. 27.

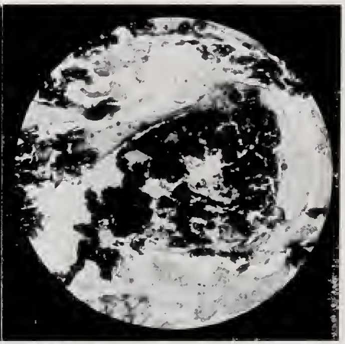


Fig. 28.

Fig. 27.—Section through the mucosa of the uterus, showing calcification of the wall of two capillaries.

Fig. 28.—Section through the foetal placenta, showing calcification. × 70.

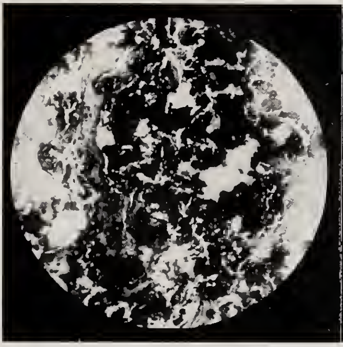


Fig. 29.

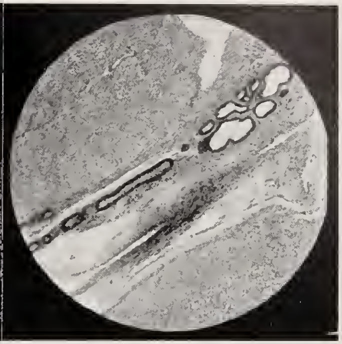


Fig. 30.

Fig. 29.—Section through foetal placenta, showing calcification. × 70.

Fig. 30.—Section through the zona muscularis of the uterus, showing heterotopia of the uterine glands. × 66.

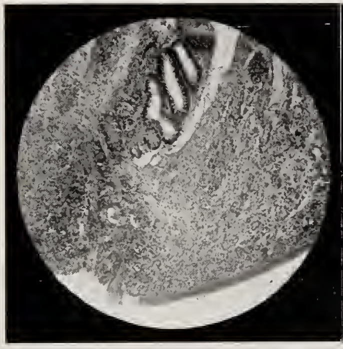


Fig. 31.

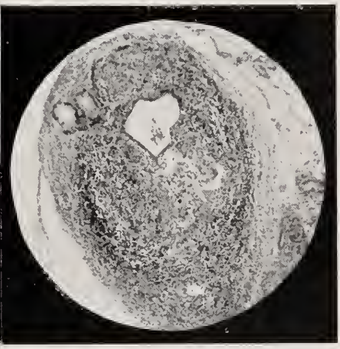


Fig. 32.

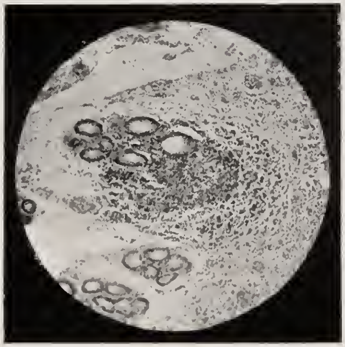
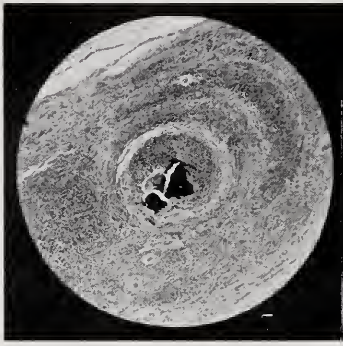


Fig. 33.

Fig. 31.—Section through the mucosa of the uterus, showing a recent tubercular nodule underneath the epithelium, which shows desquamation. × 66.

Figs. 32 and 33.—Section through the mucosa of the uterus, showing a recent tubercular nodule associated with a uterine gland. × 66.



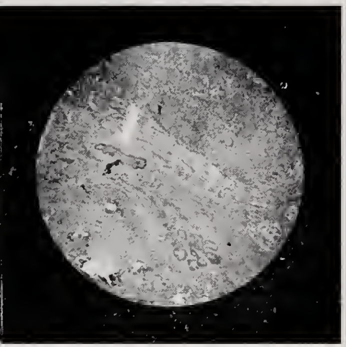
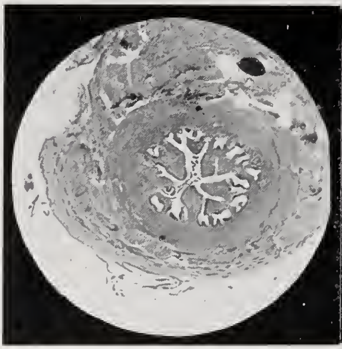


Fig. 34.

Fig. 35.

Fig. 34.—Section through the mucosa of the uterus, showing reduction in the numbers of the glands, as a result of extension of the tubercular process, and many giant cells. × 33.

Fig. 35.—Section from another portion of the same uterus as Fig 34, showing an area of calcification. × 66



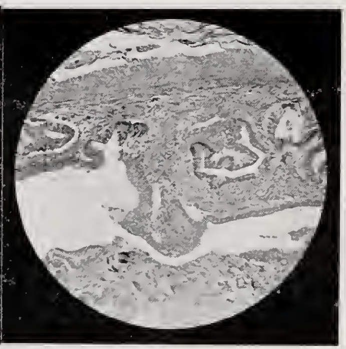


Fig. 36.

Fig. 37.

Fig. 36.—Cross-section of the fallopian tube, showing thickening of the villi with connective tissue; the epithelium is intact. × 20.

Fig. 37.—Section through the fallopian tube, showing epithelial in part desquamating, and in part complete absence of epithelium, in part normal epithelium. × 75.

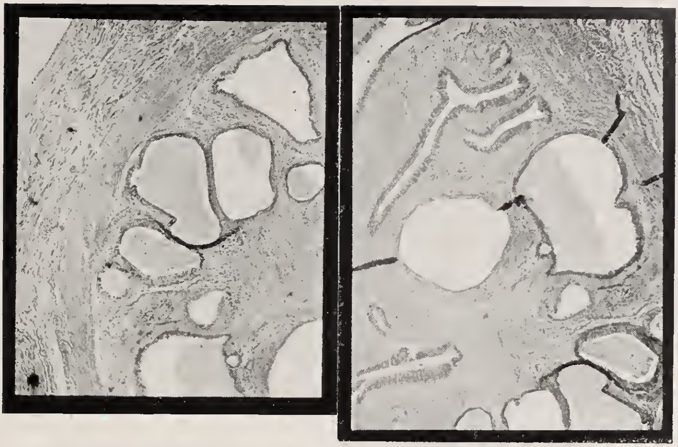


FIG. 38.　　　FIG. 39.

FIGS. 38 and 39.—Section through portion of the fallopian tube, showing marked thickening of the villi with connective tissue, and cyst-like enlargement of the crypts. The epithelium is in part cylindrical and in part flattened. × 75.

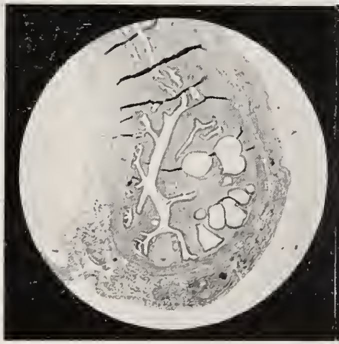


FIG. 40.

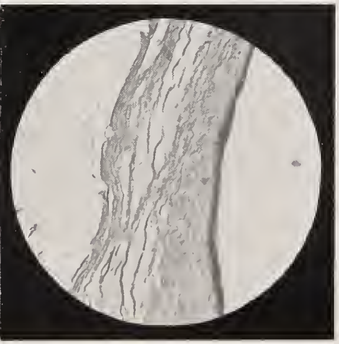


FIG. 41.

FIG. 40.—Section of tube shown in Fig. 39. × 15.

FIG. 41.—Section through the wall of the fallopian tube, showing oedema. × 40.

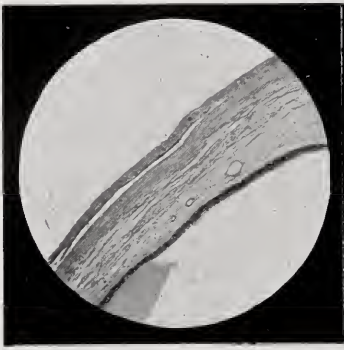


Fig. 42.

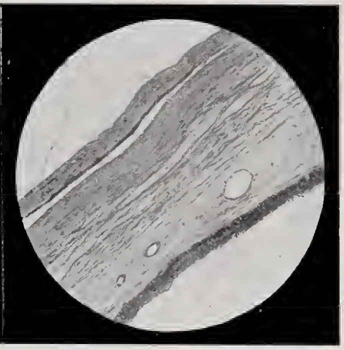


Fig. 43.

Fig. 42.—Section through the wall of the fallopian tube, showing oedema. Some of the epithelial cells show vacuolation. × 20.

Fig. 43.—Same section as shown in Fig. 42. × 40.

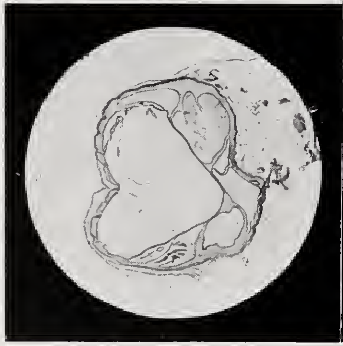


Fig. 44.

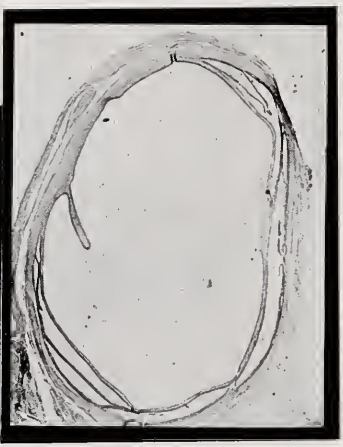


Fig. 45.

Figs. 44 and 45.—Cross-section of a fallopian tube with hydrosalpinx. 44, × 17; 45, × 45.

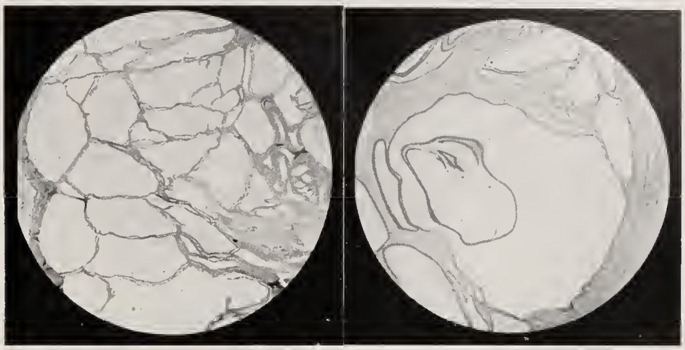


FIG. 46.

FIG. 47.

FIGS. 46 and 47.—Cross-section through portion of a fallopian tube with hydrosalpinx, showing a gland-like appearance. The septa between the vesicles are lined with epithelium. Portion of the wall is present in each figure showing atrophy. × 20.

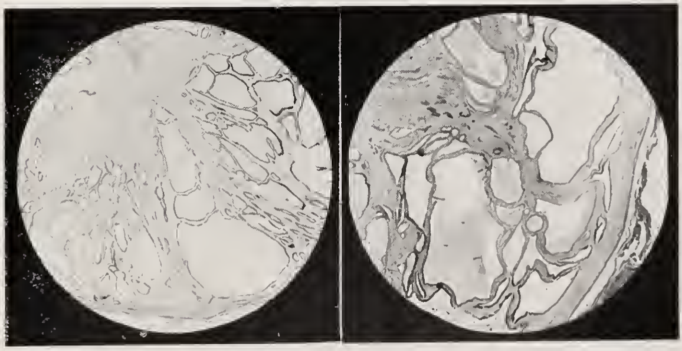


FIG. 48.

FIG. 49.

FIGS. 48 and 49.—Section through portion of a fallopian tube with hydrosalpinx, showing the villi branched and adherent towards the centre of the tube by a central connective tissue axis. 48, × 38; 49, × 20.

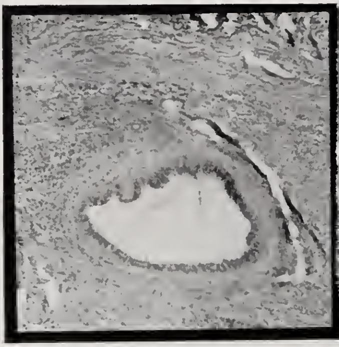


Fig. 50.

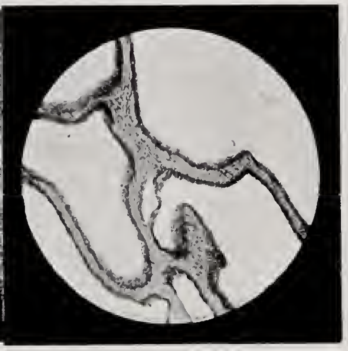


Fig. 51.

Fig. 50.—Section through the branched villi of the fallopian tube in Fig. 49, showing local epithelial proliferation. × 75.

Fig. 51.—Section through a fallopian tube with tubercular salpingitis, showing an intervillus crypt lined with cylindrical epithelium cut off and enclosed in connective tissue. × 75.

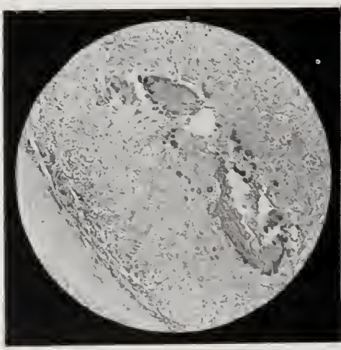


Fig. 52.

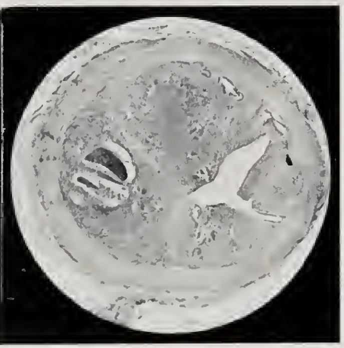
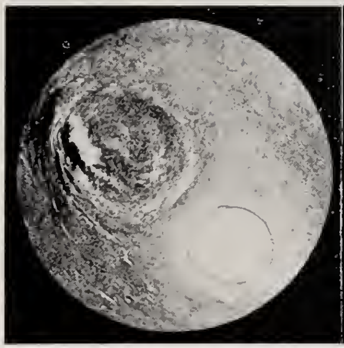


Fig. 53.

Fig. 52.—Cross-section of tubercular fallopian tube. The nodule is showing commencing necrosis. Portions of intervillus crypts are seen lined with epithelium and two are filled with cells and débris. × 20.

Fig. 53.—Section of a tubercular fallopian tube, showing calcification. A giant cell is seen close to the zona muscularis. × 75.



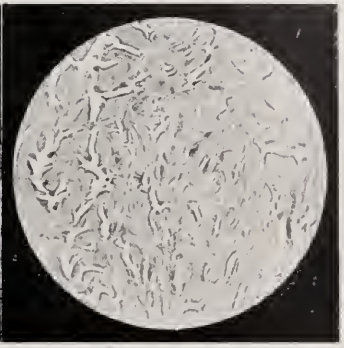
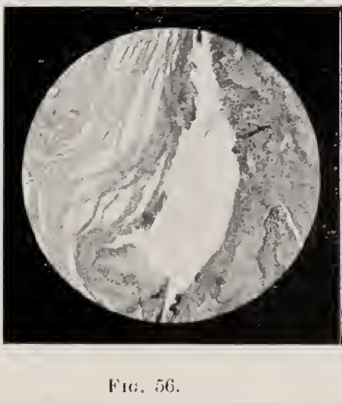


Fig. 54.

Fig. 55.

Fig. 54.—Section showing a cystadenoma of the ovary. × 45.
Fig. 55.—Section of an ovary, showing a cystic corpus albicans and an atretic follicle. × 45.



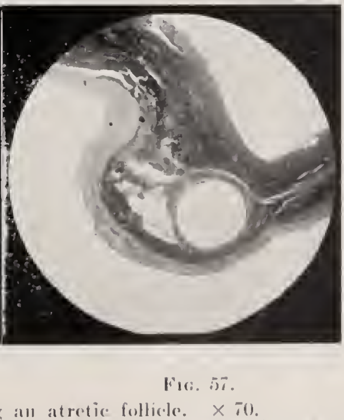


Fig. 56.

Fig. 57.

Fig. 56.—Section of an ovary, showing an atretic follicle. × 70.
Fig. 57.—Section through the rudimentary ovary of a "freemartin," showing a small cyst with a connective tissue wall. × 20.

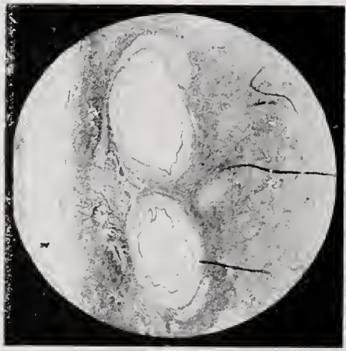


Fig. 58.

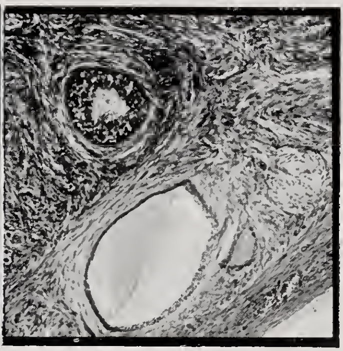


Fig. 59.

Fig. 58.—Section of an ovary, showing two small cysts with connective tissue wall and epithelial lining. The lining has become detached. × 20.

Fig. 59.—Section of an ovary, showing a small cyst lined with epithelium. × 140.

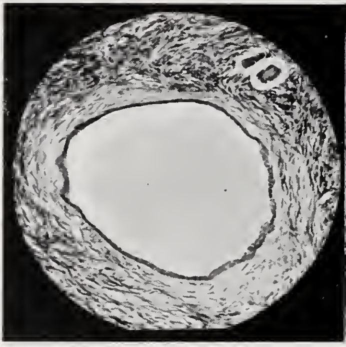


Fig. 60.

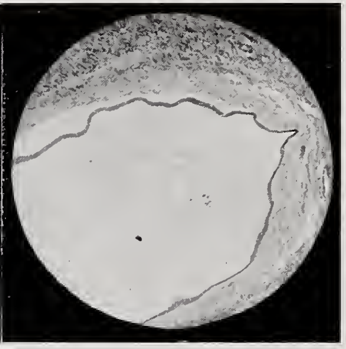


Fig. 61.

Fig. 60.—Section of an ovary, showing a similar type of cyst to that shown in Fig. 59. × 140.

Fig. 61.—Portion of a cyst shown in Fig. 58. × 70.

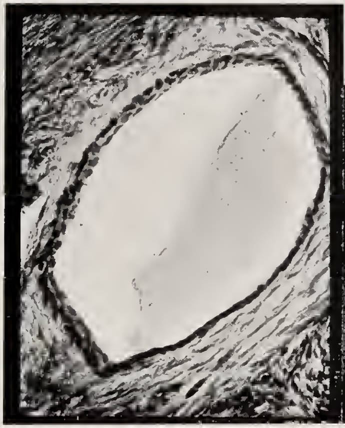


FIG. 62.

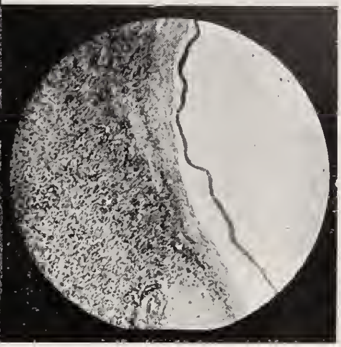


FIG. 63.

FIG. 62.—Cyst shown in Fig. 59. × 300.

FIG. 63.—Portion of the wall of a similar type of cyst as shown in Fig. 58. The epithelial lining has become detached. × 70.

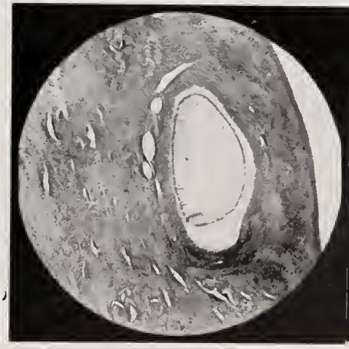


FIG. 64.

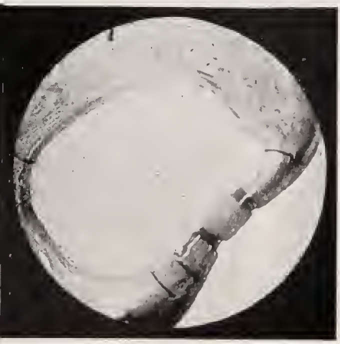


FIG. 65.

FIG. 64.—Section of the ovary, showing a small cyst. The zona granulosa has almost disappeared. The cells which remain show degeneration. The wall is composed of elements of the theca. × 20.

FIG. 65.—Section of the ovary, showing a small cyst. The wall is composed of loose connective tissue which is oedematous. × 20.

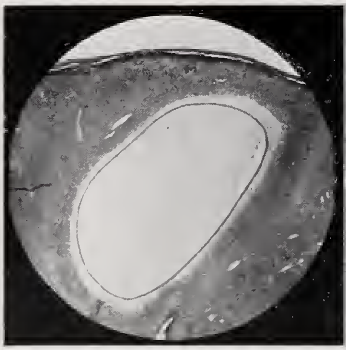


FIG. 66.

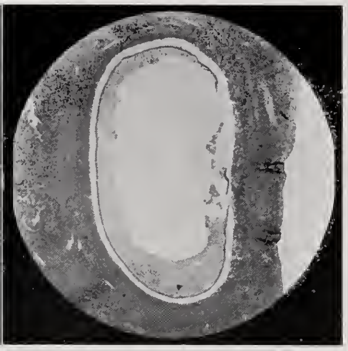


FIG. 67.

FIG. 66.—Section of the ovary, showing a small cyst. The wall is composed of loose connective tissue which is oedematous. × 20.

FIG. 67.—Section of the ovary, showing a small cyst with a connective tissue wall. A similar cyst to that shown in Figs. 65 and 66, but the wall is more dense. × 20.

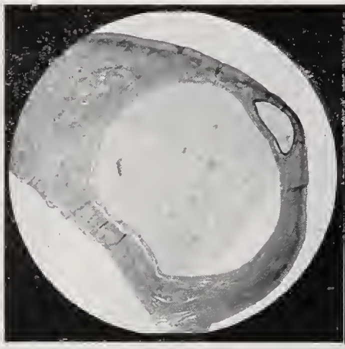


FIG. 68.

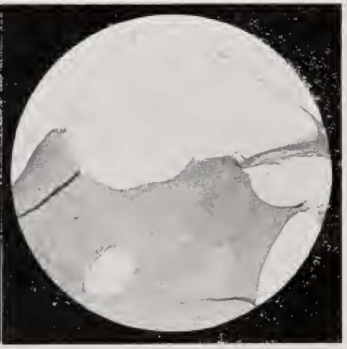


FIG. 69.

FIG. 68.—Section of the ovary, showing a small cyst with a connective tissue wall. A similar cyst to that shown in Figs. 65 and 66, but the wall is more dense. × 20.

FIG. 69.—Section showing large superficial cysts of the ovary with an extremely thin connective tissue wall which is lined with a single layer of epithelial cells. The lining is partly detached at the right and left extremities of the large cyst in the centre of the photograph. × 20.

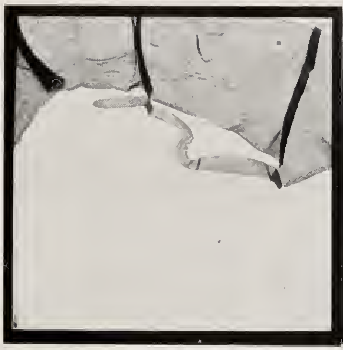


Fig. 70.

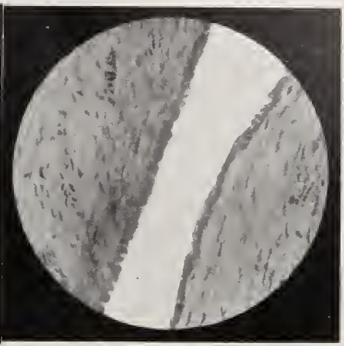


Fig. 71.

Fig. 70.—Wall of a large cyst, showing a long slender villus. It is lined with epithelium. × 17.

Fig. 71.—Section of cyst wall and villus shown in Fig. 70, showing epithelial lining. × 300.

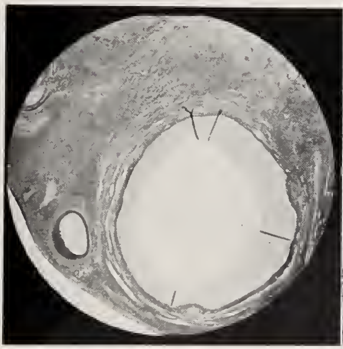


Fig. 72.

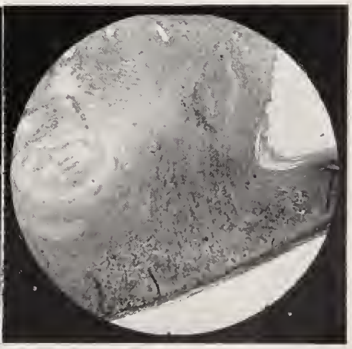


Fig. 73.

Fig. 72.—Section of the ovary, showing a cyst. The zona granulosa has disappeared. The theca is oedematous. On the left is a follicle from the wall of which portion of the follicular epithelium has become detached by manipulation. × 10.

Fig. 73.—Section of an ovary, showing portion of a cyst wall on the left and an atretic follicle on the right. × 20.

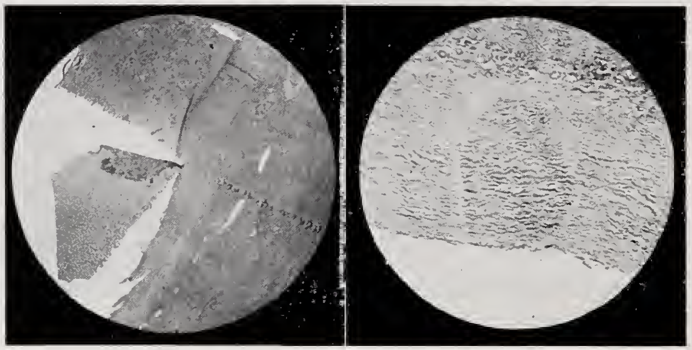


FIG. 74. FIG. 75.

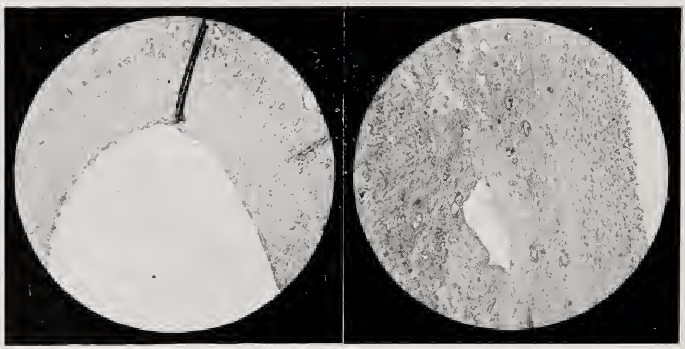


FIG. 76. FIG. 77.

FIGS. 74, 75, 76, and 77.—Section of the wall of ovarian cysts, showing the nature of the connective tissue lining. In Fig. 74 portion of the coagulated contents has been sectioned. 74, × 20; 75, × 78; 76, × 70; 77, × 78.

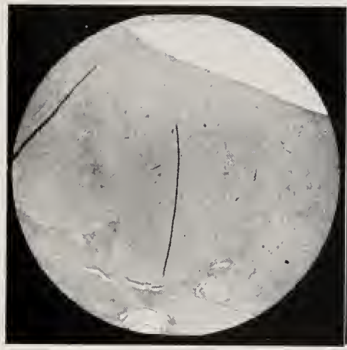


Fig. 78.

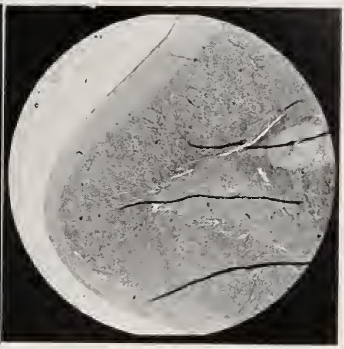


Fig. 79.

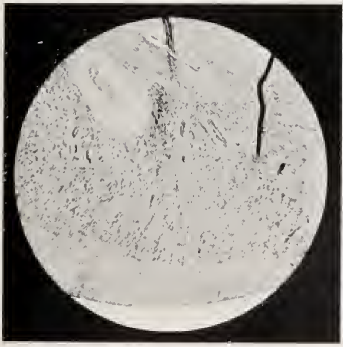


Fig. 80.

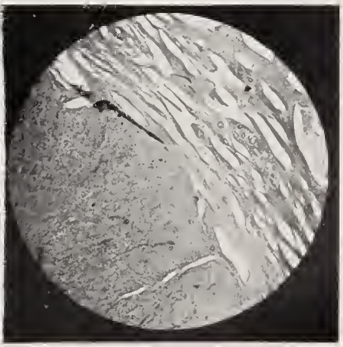


Fig. 81.

Figs. 78, 79, and 80.—Sections of the wall of lutein cysts, showing three layers: (*a*) connective tissue, (*b*) lutein tissue, (*c*) connective tissue. 78, × 10; 79, × 20; 80, × 45.

Fig. 81.—Portion of the wall of a lutein cyst, showing oedema of the outer connective tissue layer. × 20.

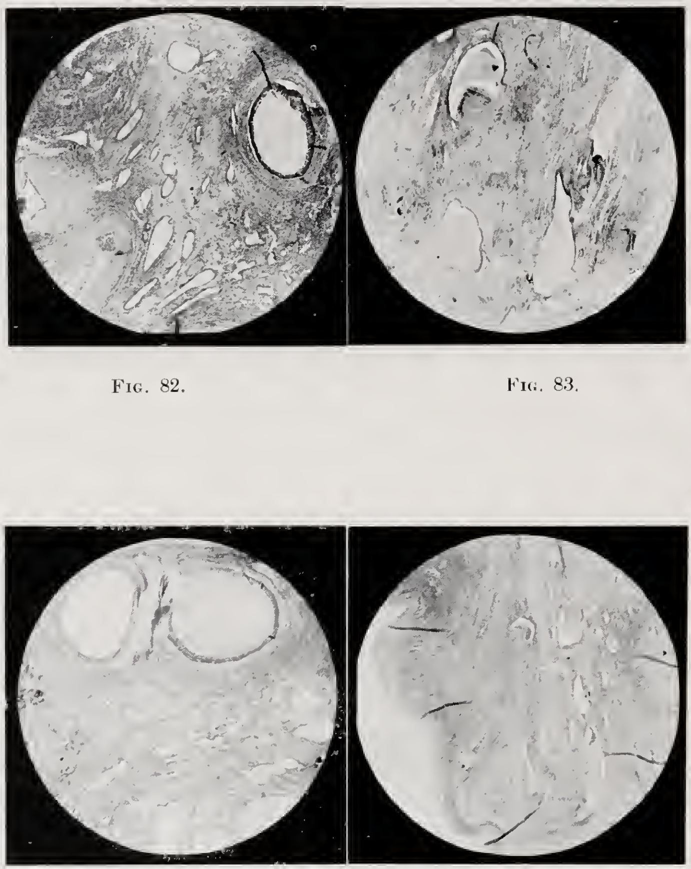


Fig. 82. Fig. 83.

Fig. 84. Fig. 85.

Figs. 82, 83, 84, and 85.—Section of the ovary, showing marked oedema. Section 82 shows a follicle from which the ovum has been removed during manipulation. Section 84 shows a similar follicle. The cyst-like structure on the left in Section 84 appears to be a follicle the granulosa of which has undergone degeneration. 82, × 20; 83, × 20; 84, × 10; 85, × 20.

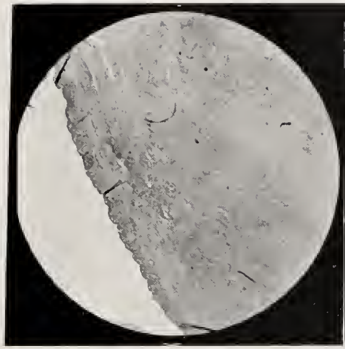


Fig. 86.

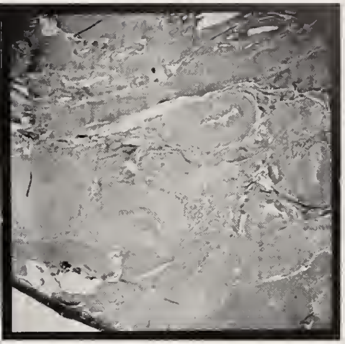


Fig. 87.

Fig. 86.—Section of an ovary, showing sclerosis. × 20.
Fig. 87.—Section of an ovary, showing hyaline degeneration of a vessel wall; several other vessels are unchanged. × 20.

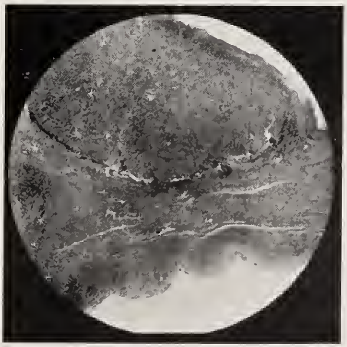


Fig. 88.

Fig. 88.—Section of an ovary, showing the wall of an old lutein cyst. Little lutein tissue remains. Towards the lower half of the photograph is seen portion of a corpus luteum from the most recent inter-ovulation period; it is also undergoing cystic degeneration. The border at the lower left extremity is seen to be composed of connective tissue which was lining the cystic cavity. × 20.

APPENDIX IV.

This Appendix shows charts of the oestral periods and duration of the inter-ovulation periods of animals suffering from sterility from various causes.

BOVINE.	No	DIAGNOSIS.	1925					1926												1927										
			Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov
	428	F.S.												▮			14 +	3 + 27 +					9 +	3 + P			8 A	3 +		D
	434	F.S.													▮			1 + P					7 A 18 +	8 + P			15 A 19 +	6 +		D
	838	F.S.									▮	24 + P																		
	839	F.S.									▮	16 + P																		
	840	F.S.									▮	23 +				9 + P														
	842	F.S.									▮	23 +	17 + P																	
	843	F.S.									▮	25 +				12 +	2 + P													
	1212	Sen. CpP.	▮				☩																							
	1213	C, M.ch.cat., CLCy., Sl., GFCy., Pa.	▮				12 +	11 +		25 +	18 + ☩																			
	1214	F.S.	▮		18 +		14 +	24 +	15 +	25 +		9 +	6 + 17 + 22 +	2 + 9 +		6 +	2 +			24 +		4 +	19 0	12 0	29 + P					
	1215	C, M.ch.cat.	▮		3 0	24 +	21 +		21 +	17 +	9 +	4 + 28 +			15 +	4 +	30 +	20 +	22 +		☩									
	1216	F.S.	▮		11 +						4 + 12 +			2 +						26 +	19 +	16 +	10 + 20 +							D

A.	ABORTED.
C.	CERVICITIS.
CL.Cy.	CORPUS LUTEUM CYST.
CpP	CORPUS LUTEUM PERSISTENS.
O.	OBSERVATIONS DISCONTINUED.
F.S.	FUNCTIONAL STERILITY.
GEN.DEF.	GENITAL DEFORMITY.
GF.Cy.	GRAAFIAN FOLLICLE CYST.
Hy.	HYDROMETRA.
M.Ch.cat.	METRITIS CHRONICA CATARRHALIS.
M.Ch.cat.Cy.	METRITIS CHRONICA CATARRHALIS CYSTICA.
NP.	NEOPLASM.
Ny.	NYMPHOMANIA.
0.	NOT SERVED.
P	PREGNANT.
Pa.	PAVILIONITIS.
Py.	PYOMETRA.
S.	SENILITY.
Sl.	SALPINGITIS.
SCyD.	SMALL CYSTIC DEGENERATION.
T.	TUBERCULOSIS.
+.	SERVED.
☩.	DEAD.
▮.	OBSERVATIONS BEGUN.

BOVINE	No	DIAGNOSIS	1925					1926												1927									
			Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct
	1217	F.S.	▮				2+		✝																				
	1218	F.S. followed by G.F.Cy.	▮			2+ 27+			15+	10+	10+	2+ 18+	8+ 13+ 19+ 21+ 25+	2+			5+		22+			7+ 29+	6+ 9+ 28+		1+ 3+ 10+ 15+	3+ ✝			
	1219	F.S. followed by G.F.Cy.	▮				9+ 31+		13+	6+	4+ 10+ 23+	1+ 5+ 9+ 17+ 23+ 21+	6+		2+ 8+				18+	7+ 14+ 22+	8+ 12+ 20+	2+ 22+ 23+ 26+	9+ 12+ 23+ 27+	6+ 11+	11+ 18+ 24+	✝			
	1220	F.S.	▮			7+ 25+	19+ ✝																						
	1228	F.S.	▮			1 0	16+			8+	13+	3+	21+	11+ P															
	1630	M.ch.cat.	▮		31+	20+			17+	27+	16+	21+ P																	
	1631	M.ch.cat., CpP., C.L.Cy.	▮ 15 0	5 0 26 0	6+ 27 0	5 0 7 0 9+ 25+	24+		8+					2+ ✝															
	1632	Hy., CpP.	▮									22+ ✝																	
	1634	M.ch.cat.	▮ 17 0	5 0 27 0	18 0	8+ P																							
	1635	F.S.	▮							18+ P																			
	1636	G.F.Cy., M.ch.cat.	▮ 27 0	17 0	8 0 30 0	19+	8+ 29+	26+					7+	12 0	✝														
	1637	GEN.DEF.	▮		✝																								
	1638	M.ch.cat., Sl.Pa.	▮			9+		18+	1+ 22+	✝																			

A.	ABORTED.	GEN.DEF.	GENITAL DEFORMITY.	Ny.	NYMPHOMANIA.	Sl.	SALPINGITIS.
C.	CERVICITIS.	G.F.Cy.	GRAAFIAN FOLLICLE CYST.	O.	NOT SERVED.	S.Cy.D.	SMALL CYSTIC DEGENERA
C.L.Cy.	CORPUS LUTEUM CYST.	Hy.	HYDROMETRA.	P	PREGNANT.	T.	TUBERCULOSIS.
CpP	CORPUS LUTEUM PERSISTENS.	M.Ch.cat.	METRITIS CHRONICA CATARRHALIS.	Pa.	PAVILIONITIS.	+.	SERVED.
D	OBSERVATIONS DISCONTINUED.	M.Ch.cat.Cy.	METRITIS CHRONICA CATARRHALIS CYSTICA.	Py.	PYOMETRA.	✝.	DEAD.
FS.	FUNCTIONAL STERILITY.	NP.	NEOPLASM.	S.	SENILITY	▮.	OBSERVATIONS BEGUN.

BOVINE	No	DIAGNOSIS	1925					1926												1927	
			Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb
	1639	M.ch.cat.Cy.,C.F.Cy.	■			7 21 + +		15 +		✝											
	1657	C.Mch.cat.,Sl.,NP.,CL.Cy.	■ 27 0		31 0	25 +	5 28 + +	18 +	15 +	16 + ✝											
	1658	C.,Py.,C.F.Cy.	■		30 0		4 30 + +	17 +	8 +	17 +			16 21 0 0	29 0 ✝							
	1659	Mch.cat.,Sl.,CL.Cy.	■			7 +	6 +			13 +	9 + ✝										
	1660	T.	■	11 0	27 0	6 22 0 +	11 +	2 21 + +	8 26 + +	18 +	7 +	1 19 0 +		20 0 ✝							
	1661	Mch.cat.	■	17 0	26 0	9 23 30 + + +	P														
	1662	M.ch.cat., followed by Py.& CL.Cy.	■			16 +	15 30 + +		1 +	1 20 + +	11 0	3 0 ✝									
	1663	Mch.cat.Cy.,C.F.Cy.	■ 26 0									21 +					14 +				✝
	1664	Mch.cat.Cy.,C.,Sl.	■	5 26 0 0	25 0	13 29 + +	26 0	26 0				20 + ✝									
	1665	Mch.cat.,CpP.	■ 26 0				9 +		20 +	16 +	6 +	1 20 + +			✝						
	1684	Mch.cat.	■			10 +	4 + P														
	1685	Mch.cat.,Pa.,CpP.	■		30 0				13 +		11 + ✝										

BOVINE	No	DIAGNOSIS	1925					1926												1927										
			Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov
	1686	Mch.cat.Pa.Sl.CL.Cy.	▮			15 +	4 +				20 0 †																			
	1691	M.ch.cat.	▮	17 0	9 0	6 23 0 +			22 +	15 +	4 +	16 +	9 +	1 +	29 +	19 +	13 +	3 22 + +		9 13 31 + + +	P									
	1805	T.	▮		31 0 †																									
	1807	T.	▮		26 0 †																									
	1870	Mch.cat.Sl.Pa. GF.Cy. GEN.DEF.						▮	7 +	24 +		18 0	10 24 0 0	†																
	1871	Mch.cat.Sl.Pa.						▮ 8 29 + +		14 +	7 29 + 0	†																		
	1886	GEN.DEF.						▮																						D
	2071	Py.CpP.									▮		†																	
	2073	Mch.cat.Sl.CL.Cy.									▮	2 26 0 +	†																	
	2074	Mch.cat.C.Sl.CL.Cy.									▮		†																	
	2109	F.S.												▮ 14 0	2 25 0 +	15 0	28 +			4 +				10 27 + +	17 +	9 31 + +	21 +	14 +	6 +	D
	2110	Mch.cat.Sl.Pa.CL.Cy.												▮ 15 0	4 26 0 0	16 0 †														

A.	ABORTED.	GEN.DEF.	GENITAL DEFORMITY.	Ny.	NYMPHOMANIA.	Sl.	SALPINGITIS.
C.	CERVICITIS.	GF.Cy	GRAAFIAN FOLLICLE CYST.	0.	NOT SERVED.	SCyD.	SMALL CYSTIC DEGENERATION
CL.Cy	CORPUS LUTEUM CYST.	Hy	HYDROMETRA.	P	PREGNANT.	T	TUBERCULOSIS.
CpP	CORPUS LUTEUM PERSISTENS.	M.Ch.cat	METRITIS CHRONICA CATARRHALIS.	Pa	PAVILIONITIS.	+.	SERVED.
D.	OBSERVATIONS DISCONTINUED.	M.Ch.cat.Cy	METRITIS CHRONICA CATARRHALIS CYSTICA.	Py	PYOMETRA.	†.	DEAD.
F.S.	FUNCTIONAL STERILITY.	NP	NEOPLASM.	S.	SENILITY.	▮.	OBSERVATIONS BEGUN.

BOVINE	No	DIAGNOSIS	1926						1927										
			July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept	Oct	Nov
	2111	Mch.cat., Sl.	▮	25 0	✝														
	2112	Mch.cat., Sl. Pa.	▮ 16 0	5 25 0 0	✝														
	2113	Mch.cat., Sl. GF.Cy.	▮ 30 0	28 0			26 0			10 0				7 +	22 +	10 +	18 +		D ✝
	2114	Mch.cat. Sl. Pa. CL.Cy.	▮ 28 0	17 0 ✝															
	2115	Mch.cat. Sl. Pa. CL.Cy.	▮ 22 0	11 0			20 24 + 0	✝											
	2141	GEN. DEF.		▮		✝													
	2142	GEN. DEF.		▮			✝												
	2143	Mch.cat.Cy., GF.Cy.		▮		✝													
	2144	Mch.cat.Cy., GF.Cy.		▮	16 0	7 30 0 0					19 0	8 29 0 0					20 +	13 +	D
	2145	Mch.cat.Cy., GF.Cy.		▮				22 0	13 0		25 0						3 27 + +		
	2146	Mch.cat., C.		▮ 27 0	21 0		✝												
	2147	Mch.cat.		▮	24 0	18 0	29 0		12 0		8 +	9 +	7 31 + +	24 +	18 +				D

BOVINE	No	DIAGNOSIS	1926					1927								
			Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apl	May	June	July	Aug	Sept
	2148	F.S.	▮	17 0					15 0				29 0		✝	
	2149	Mch.cat.,C.,GF.Cy.	▮ 28 0		7 0 ✝											
	2150	Mch.cat.,C.,C.L.Cy.	▮	25 0	✝											
	2159	Mch.cat.,CpP.	▮									22 0	14 0	7 +	✝	
	2160	Mch.cat.Cy.,Sl.,C.L.Cy.	▮		5 0		7 0	5 0	20 0	31 0	20 0		3 0	24 +	30 +	✝
	2161	Mch.cat.,GF.Cy.,	▮	6 0						2 28 0 0	15 29 0 0	11 0	1 24 0 0		✝	
	2162	Mch.cat.,Sl.,Pa.	▮		27 0	20 0	14 0	✝								
	2163	Py.		▮												✝
	2264	Mch.cat.Cy.,C.,NP.						▮	✝							

APPENDIX V.

In this Appendix are shown the dimensions and weights of the epiphysis, hypophysis, thyroid, and adrenal glands in the sterile cows slaughtered, as compared with the dimensions and weights of these glands in normal cattle killed at the Pretoria Abattoir.

SIZE AND WEIGHT OF ENDOCRINES OF STERILITY CATTLE.

No. of Bovine.	Epiphysis.				Hypophysis.				Adrenals.								Thyroid.						Thyroid, including Isthmus.	
									Right.				Left.				Right Half.			Left Half.				
	L. cm.	B. cm.	W. cm.	Wgt. gm.	L. cm.	B. cm.	W. cm.	Wgt. gm.	L. cm.	B. cm.	W. cm.	Wgt. gm.	L. cm.	B. cm.	W. cm.	Wgt. gm.	L. cm.	B. cm.	W. cm.	L. cm.	B. cm.	W. cm.	L. cm.	Wgt. gm.
1212	—	—	—	—	—	—	—	—	5.0	5.0	2.0	20.0	7·5	4.0	1·5	19.0	4·5	7.0	1.0	5.0	6·5	1.0	18·5	—
1213	1·3	0·7	0·6	0·3	2·7	2·8	2·8	4·02	8·2	4.0	1.0	21.0	5.0	5.0	1·5	19.0	4·8	9·8	1.0	4·8	7·0	0·9	19·5	47.0
1293	—	—	—	—	—	—	—	—	4·5	2·5	1.25	8.0	4.0	3.0	1·25	7.0	3·5	4·0	—	3·5	3·5	—	17·0	—
1631	1·2	0·3	0·3	0·2	1·8	1·5	1·3	3·5	6·2	3·8	1·2	15·5	4·3	4.0	1·8	15.0	6.0	3.0	1·0	6·0	3·5	1·2	25·0	25.0
1632	1·3	0·4	0·4	0·3	2·6	1·7	1.0	3.0	6·9	3·5	1·3	15·0	5·2	3·8	1·6	13·5	5·7	6·2	0·6	4·5	3·7	0·8	23·0	21·5
1633	—	—	—	—	—	—	—	—	6·0	3·5	1·0	15·5	4·0	4·0	1·5	12·5	3·5	7·0	1·0	3·5	6·0	1·0	21·0	26·5
1636	1·5	0·4	0·4	0·15	2·0	1·8	1·2	2·5	4·2	4·6	1·9	14·0	5·5	3·2	1·5	17·0	4·8	5·5	0·6	5·6	3·0	1·0	27·0	20·5
1637	—	—	—	—	2·75	2·0	1·0	2·2	5·75	4·0	1·25	13·0	5·0	3·5	1·25	12·0	4·0	5·0	—	4·0	4·5	—	19·5	22·5
1638	1·7	0·7	0·7	0·35	2·1	1·6	1·5	2·8	6·3	4·2	1·0	19·5	5·0	4·8	1·3	18·0	4·0	6·0	0·8	3·7	6·0	0·8	23·0	24·5
1639	—	—	—	0·2	2·0	1·6	1·6	3·8	8·0	5·0	1·6	40·9	5·9	5·6	1·7	38·2	—	9·0	1·4	—	9·0	1·0	25·0	87·5
1640	1·5	0·6	0·6	0·3	2·7	2·4	1·5	3·75	7·0	5·4	2·5	35·5	7·0	5·8	2·2	27·75	5·0	7·0	—	4·5	6·0	—	23·0	39·5
1657	1·3	0·4	0·5	0·2	2·2	1·1	1·7	2·4	5·4	4·3	1·2	13·0	4·4	5·5	1·4	12·0	4·6	4·8	1·2	6·6	6·4	1·0	19·5	23·5
1658	1·3	0·4	0·4	0·3	2·1	1·8	1·0	2·7	6·8	4·0	1·7	20·5	6·0	3·3	1·5	20·0	5·0	5·0	1·4	7·0	5·0	1·2	24·0	30·5
1659	1·7	0·8	0·6	0·42	2·0	1·6	1·5	3·0	7·0	4·0	1·3	20·0	6·2	5·0	1·3	16·5	4·8	6·4	0·7	6·0	6·3	0·8	31·8	30·0
1660	1·4	0·7	0·7	0·4	2·4	1·8	1·4	3·5	5·0	3·6	1·6	14·5	4·6	2·8	1·5	11·0	4·0	4·5	1·0	5·0	2·6	0·9	21·2	20·0
1662	1·4	0·5	0·6	0·3	2·5	1·7	1·6	3·0	6·0	3·7	0·8	12·0	4·5	4·4	1·1	13·0	4·5	4·4	0·8	5·0	3·2	0·6	22·0	14·5
1664	1·8	0·6	0·6	0·45	2·2	1·5	1·3	2·5	6·1	3·6	1·3	17·5	5·1	4·4	1·5	16·0	4·8	3·7	1·2	3·8	4·7	1·0	22·2	22·5
1683	—	—	—	—	—	—	—	—	5·4	3·75	1·25	14·0	4·75	4·25	1·25	12·75	4·5	5·0	0·75	5·5	4·5	0·75	21·0	22·0
1685	1·4	0·7	0·7	0·3	2·0	2·5	1·4	3·5	5·5	4·2	2·0	19·5	5·0	4·8	1·8	15·0	4·5	5·0	0·8	4·8	4·6	0·8	23·5	23·0
1686	1·5	0·5	0·5	0·3	2·0	1·6	1·0	2·0	5·8	4·6	1·6	14·5	4·8	4·4	1·5	14·0	4·5	4·4	1·2	6·0	3·5	0·9	23·5	24·5
1806	—	—	—	—	—	—	—	—	5·75	3·75	1·5	17·5	4·5	4·0	1·25	16·0	5·0	5·0	0·6	5·0	4·25	0·6	20·5	20·0
1807	—	—	—	—	2·6	1·5	1·7	4·6	7·2	4·3	1·5	25·5	4·7	5·0	2·0	22·7	5·8	5·5	1·0	5·2	6·0	0·8	20·0	20·6
1808	—	—	—	—	—	—	—	—	7·0	4·0	1·25	16·25	5·5	4·0	1·5	15·2	5·5	4·5	—	4·5	6·0	—	22·0	24·0
1813	1·0	0·7	0·5	0·4	2·4	2·2	1·4	5·0	8·2	4·6	1·8	28·5	5·6	4·9	1·8	29·0	7·0	6·5	1·3	8·0	6·4	1·2	28·0	51·5
1870	1·2	0·6	0·6	0·3	2·3	1·8	1·3	3·0	8·0	3·1	1·3	15·0	4·5	3·8	1·1	14·0	7·4	4·5	1·2	6·0	4·2	1·1	21·0	34·5
1871	1·5	0·5	0·5	0·3	2·0	1·5	1·0	2·0	5·0	3·6	1·3	13·0	4·2	4·0	1·4	11·0	8·0	3·2	1·2	5·4	3·0	0·9	22·0	27·0
2071	1·1	0·5	0·3	0·2	2·2	1·8	0·9	2·5	6·2	4·3	1·4	17·5	4·3	4·2	1·6	15·5	4·5	6·0	0·7	5·2	3·5	0·9	24·2	25·0
2073	1·2	0·4	0·4	0·15	2·5	1·6	1·3	3·0	4·2	5·5	1·2	14·5	4·7	4·0	1·1	13·5	4·5	4·8	1·0	4·4	3·8	1·0	29·2	24·5
2074	1·5	0·5	0·5	0·4	2·4	1·6	1·2	3·0	6·5	4·5	1·4	18·5	5·8	3·0	1·3	13·5	4·8	5·6	0·6	5·0	3·5	0·8	21·0	13·5
2110	1·5	0·6	0·6	0·2	2·5	1·8	1·2	3·0	4·8	3·9	1·2	11·5	6·1	3·5	1·3	12·7	5·5	5·2	1·2	5·0	4·3	1·2	19·0	25·5
2111	1·2	0·5	0·5	0·5	2·2	1·5	1·5	3·0	4·5	4·5	1·4	13·5	5·5	4·9	1·2	16·0	5·0	4·0	0·9	5·0	4·7	1·0	19·0	22·5

2112	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
2114	1·3	0·4	0·3	0·15	2·4	1·4	1·5	2·75	5·8	4·2	0·8	15·7	4·7	3·7	1·5	12·35	6·0	3·7	0·9	6·4	4·5	1·0	20·9	37·0
2141	1·4	0·6	0·6	0·3	1·8	1·6	1·4	3·0	3·2	2·8	1·5	6·5	5·5	3·0	1·4	9·0	4·0	3·5	1·0	3·8	2·5	0·8	18·8	14·5
2143	1·3	0·4	0·4	0·2	2·4	1·8	1·3	3·5	4·0	3·0	1·3	9·0	4·6	3·0	1·2	10·0	5·3	4·6	1·3	6·5	5·0	1·0	26·0	32·0
2149	1·2	0·5	0·5	0·2	2·8	2·6	1·6	8·5	6·7	4·0	2·0	23·5	5·0	5·2	2·2	20·0	8·0	7·0	1·0	8·0	7·5	1·5	30·5	53·5
2150	1·4	0·8	0·7	0·35	2·5	1·8	1·8	5·0	5·3	4·0	1·5	16·5	6·7	3·3	1·6	18·0	5·5	5·0	1·0	5·5	4·6	1·3	20·0	26·5
2142	1·2	0·5	0·5	0·2	2·0	1·2	1·4	2·5	3·6	2·8	1·2	6·5	4·4	3·1	0·8	7·5	4·8	3·6	1·5	4·0	3·4	1·2	18·4	18·5
2146	1·2	0·7	0·7	0·3	2·8	1·7	1·4	3·5	6·5	4·0	1·8	20·0	4·5	4·0	2·0	19·5	5·0	6·8	1·3	5·0	6·0	1·2	22·0	38·0
2115	1·4	0·4	0·4	0·3	2·5	1·5	1·5	3·0	6·0	4·2	1·2	13·0	5·0	3·2	1·2	12·0	5·2	5·0	1·2	5·0	3·8	0·8	19·0	22·5
2264	1·3	0·5	0·5	0·35	2·4	2·0	1·6	4·5	5·5	4·0	1·4	19·0	4·5	4·0	1·5	18·5	5·5	4·8	1·3	6·0	5·5	1·3	25·0	35·5
1663	1·6	0·5	0·5	0·6	2·6	2·0	1·4	4·5	6·8	2·6	1·2	17·5	4·7	4·0	1·4	20·0	5·0	3·2	1·0	4·5	3·5	1·0	22·0	18·5
1215	1·7	0·7	0·7	0·4	2·6	1·8	1·6	5·0	5·0	4·5	1·0	11·0	6·5	3·8	1·6	17·0	6·0	4·0	1·0	6·0	4·2	1·2	24·0	29·0
1218	1·5	0·6	0·6	0·7	2·0	0·7	0·6	4·0	6·5	3·4	1·3	13·0	4·5	4·2	1·4	10·5	6·0	5·0	1·4	5·0	5·0	1·3	21·0	33·0
1219	1·5	0·6	0·6	0·5	2·6	2·0	1·6	3·3	6·5	3·8	1·2	15·5	4·6	4·0	2·0	13·5	6·0	4·5	1·8	8·0	4·5	2·2	20·0	49·5
1665	2·0	0·75	0·75	0·75	2·5	2·0	1·25	3·0	5·2	3·2	1·25	14·5	4·5	4·25	1·5	16·0	8·0	11·0	0·75	7·0	6·0	0·75	23·5	36·0
2148	1·6	0·75	0·75	0·75	2·5	2·5	1·5	4·0	5·0	4·5	1·5	21·5	6·5	3·5	1·5	21·5	8·0	5·8	1·5	7·0	6·5	1·0	25·5	46·5
2161	1·25	0·5	0·5	0·4	2·0	1·5	1·25	3·5	4·5	4·0	1·25	12·5	6·0	3·5	1·0	14·0	5·0	4·0	1·0	5·0	4·5	1·0	29·5	22·5
2160	1·3	0·4	0·4	0·2	2·7	1·5	1·7	3·75	6·0	3·8	1·4	14·5	6·0	3·0	1·8	12·5	6·0	6·0	1·0	7·0	3·5	1·0	23·0	30·5
2163	1·25	0·7	0·7	0·75	2·4	1·7	1·3	4·0	5·0	3·5	1·5	16·0	5·5	3·5	1·7	20·0	6·0	5·0	0·6	5·5	3·5	0·7	23·5	28·0
2113	1·5	0·6	0·6	0·4	2·0	1·8	1·3	2·5	5·5	4·0	1·4	12·5	4·0	3·3	1·5	9·0	7·5	5·0	1·0	6·0	3·2	1·0	20·0	25·5
2147	1·4	0·5	0·5	0·3	2·5	1·8	1·4	4·0	7·3	3·0	1·6	15·0	4·5	4·2	2·0	17·5	5·0	5·0	1·2	5·0	4·5	0·9	20·2	29·0
Averages for S.A. Sterile Cows	1·39	0·55	0·54	0·35	2·33	1·75	1·39	3·41	6·0	3·96	1·4	16·64	5·13	4·01	1·49	15·8	5·37	5·31	1·03	5·42	4·69	1·0	23·05	29·23
Averages for Normal S.A. Cattle	1·6	0·5	0·5	0·35	2·4	1·73	1·3	3·26	4·8	3·46	1·16	11·0	5·3	3·11	1·2	10·65	4·17	3·69	1·02	4·58	3·35	0·95	20·22	16·1
Weights, Measurements, from Joest	1–2·0	0·5–0·8	0·5–0·8	0·2–0·3	2·0–2·6	1·6–1·9	1·5–1·6	1·9–4	5·0	3·5	2·0	16·0–17·0	5·0	3·5	2·0	16·0–17·0	6·7	4·0–5·0	0·75–1·5	6·0–7·0	4·0–5·0	0·75–1·5	—	21·0–36·0

Section VIII.

Mineral Deficiency.

P. J. du Toit and J. H. R. Bisschop. The Breeding of Cattle on Phosphorus Deficient Veld.

15th Annual Report of the Director of Veterinary Services, Union of South Africa. October, 1929.

The Breeding of Cattle on Phosphorus Deficient Veld.

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and

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VII. SUMMARY AND CNCLUSIONS.

I. INTRODUCTION.

THE significance of phosphorus as a factor in the health of cattle was first realized in South Africa about 10 years ago in the course of investigations into " Lamsiekte " or Parabotulism of cattle. With the progress of this work, it became more and more clear that phosphorus and other minerals play a very important rôle, not only in the health of the animals, but also in their development and propagation.

This "newer knowledge" of nutrition is now claiming the attention of scientists all over the world. Dr. J. B. Orr, Director of the Rowett Research Institute, Aberdeen, and one of the foremost workers in this field, has recently compiled a most useful treatise on the subject of "Minerals in Pasture and their Relation to Animal Nutrition," and a few quotations from the Foreword* to this work may best serve to introduce the subject matter of the present paper.

The Science of Animal Nutrition has made enormous progress in the course of the present century, but "the aspect of the problem to which most attention was devoted was that simply of balance of intake and output, either stated in terms of energy, or in terms of absolute amounts of protein, carbohydrates, and fat."

"Concentrated work on a wide front in animal nutrition has led many investigators . . . to the conclusion that in the nutrition of farm animals substances accessory to the energy-yielding materials of a diet are of prime importance and that their absence leads to deficiency diseases . . . not only amongst stall-fed animals, but also amongst grazing animals, ranging freely over wide stretches of country."

Although "it has long been known that various mineral constituents like Calcium, Phosphorus, Iron, Iodine . . . were as essential for the life of the organism as the energy giving foodstuffs themselves, their true significance in the animal organism, is not even yet perhaps fully appreciated." This field of nutritional physiology is "practically untilled . . . and the economic possibilities are immense."

It will be obvious, from a perusal of the following pages, that the "economic possibilities" of phosphorus feeding to cattle in the phosphorus deficient areas of South Africa are indeed "immense." In earlier publications Theiler, Green, and Du Toit (1924 and 1927) have demonstrated the great value of phosphorus feeding to the individual animals. The present paper is intended to prove that phosphorus is indispensable in the process of breeding better-class stock. Indeed, it may be said that, without phosphorus feeding, many parts of South Africa will never carry anything but scrub cattle, and even these will gradually die out; whereas with the addition of a small ration of phosphorus to the ordinary veld-grazing, there is no reason why those same areas should not produce cattle of the best breeds and highest quality.

The scope of the present paper was indicated in a short preliminary note by Theiler, Green, and Du Toit (1928). In the meantime Sir Arnold Theiler has retired from the Service and Dr. H. H. Green has also left South Africa. The work was continued under the control of the Director of Veterinary Services and with the collaboration of Mr. J. H. R. Bisschop, who during the period July, 1927, till August, 1928, was in charge of the Experiment Station at Armoedsvlakte and supervised the experiments personally. Since August, 1928, Mr. J. G. Bekker has been in charge of the Station, and the authors wish to express their gratitude to him and the rest of the staff at Armoedsvlakte for the meticulous care with which the experiments have been carried out and for their devotion to the work generally.

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* By Prof. E. P. Cathcart.

II. BRIEF HISTORICAL SURVEY OF EVENTS LEADING UP TO THE PRESENT INVESTIGATION.

In their comprehensive report on Lamsiekte (Parabotulism) of cattle in South Africa, Theiler, Viljoen, Green, Du Toit, Meier, and Robinson (1927) give a detailed historical account of the disease, from the 18th century down to the time (1919) when its etiology was cleared up.

For the purposes of the present paper, it is unnecessary to recount those historical data. However, brief references may be made to some of the views held at different times and by different authors about the nature and cause of the diseases Lamsiekte and Styfsiekte.

In the early stages, the symptoms of these two diseases are very similar "lamsiekte being characterized by paralysis and paresis, principally of the locomotory system," while styfsiekte " is a highly congested, sometimes even inflamed, condition of the ends of the long bones which form the joints of the limbs, principally of the fore legs, and accompanied by acute pain and lameness." (Theiler, 1912.)

The first scientific observations on these diseases were made by Hutcheon, Colonial Veterinary Surgeon in the Cape, in whose publications we find references from 1884 to 1903.

" Hutcheon correlated the diseases with the ' perverted appetite ' shown by cattle in lamsiekte and styfsiekte areas, and regarded the diseases as due to deficiency of lime and phosphorus in the soil."

Some of his observations may be quoted here as being of special interest to the subject under discussion.

> As long as memory can serve, the diseases have been known in Bechuanaland.
>
> Even in olden times the prevalence of the disease was associated with dry years and dry seasons.
>
> It was well known that styfsiekte was found prevalent in the same areas where lamsiekte existed.
>
> Where styfsiekte and lamsiekte occurred, cattle had a craving for rotten bones.
>
> The increasing prevalence of styfsiekte in 1882 was considered to be due to the dry season.
>
> All classes of stock are susceptible, but more particularly growing stock and cows in calf or giving milk. Full-grown oxen were seldom affected.
>
> Cows before and immediately after calving are more subject than the same animals at other times.
>
> The diseases were found on different kinds of soil, and all these soils showed clear indications of lack of phosphates.
>
> When stock are supplied with mealie stalks, chaff, bran, etc., the disease disappeared.
>
> Cattle contracted the diseases more easily on certain parts of the farm than on others.
>
> Healthy cattle, freshly introduced from a farm where the diseases did not prevail, did not contract them as quickly as did native stock.

Hutcheon came to the conclusion " that lamsiekte and styfsiekte were two different forms of one and the same disease: the common factor in both, as a primary cause, being a want of phosphates in the system. This want tended to produce styfsiekte, but, when another exciting cause, possibly a plant, was present, lamsiekte might follow." (Theiler, 1912.)

This view received strong support from the work of Borthwick in 1895, who showed that regular feeding of large quantities of bonemeal greatly reduced the incidence of the disease. It also seemed to be supported by the soil analyses of Juritz, which revealed a widespread deficiency of phosphorus in the soils of South Africa.

Theiler, in 1912, gave the results of his investigations and observations, and of information obtained from farmers in reply to queries sent out, and from interviews and correspondence, in the form of an interesting and very valuable article.

Many of his conclusions are again of special interest to the subject of our inquiry, and may be quoted here:—

> " In the Humansdorp district, the disease is generally dated back to the time of the big fire, in the sixties, which raged from George to Uitenhage. As a result of this fire, it is stated that sour bush invaded the pastures.
>
> " In the Riversdale and Bredasdorp areas, the diseases occur only on sour veld and never on sweet veld.
>
> " It has been stated that no case of lamsiekte has ever occurred on Karoo (sweet) veld, although cattle which were moved in have died.
>
> " It is stated by many observers that cattle born and bred on a farm are more liable to contract the diseases. Cattle introduced from elsewhere seem to be free of them for some time after their arrival. After a lapse of twelve months, there seems to be no difference.
>
> " Lamsiekte and styfsiekte principally attack heifers in calf and cows with their first, second, or third calf. Cows in milk are said to be more susceptible than cows and heifers in calf.
>
> " On lamsiekte farms cattle show an unusual craving for bones or anything abnormal, such as skins, leather, tins, hoop iron, fencing wire, manure, horns, clothing, grain bags, earth, bark of trees, limestone, etc. Where cattle do not crave for bones, as in the Karroo, they do not die of lamsiekte.
>
> " A craving for bones seems to be found in cattle which are grazing on sour veld. This craving appears when cattle are brought from sweet to sour veld, and disappears when they are brought from sour to sweet veld. When veld is poor there are more cravers than when the veld is good.
>
> " Farmers who came to Bechuanaland from other parts of South Africa state that craving is more noticeable amongst cattle there than anywhere else in South Africa.
>
> " The diseases are said to occur more extensively in lime and dolomite formations and more on shallow than on deep soils.
>
> " The diseases are more prevalent in dry years than in years of normal rainfall.
>
> " The majority of farmers believe that the feeding of bonemeal in no way influences lamsiekte, while a small minority claim a protective influence. Practically all farmers admit a remarkable influence, due to bonemeal, on the condition of an animal."

Theiler, in the same article, discussed the various theories, postulated at the time (1912) as to the cause of lamsiekte.

He admits that the " Want of Nutrition Theory " as postulated by Hutcheon, explains satisfactorily many factors associated with the etiology of the disease, and that the phosphorus deficiency may set up the disease, but the theory cannot explain certain very important associated factors.

The theory broke down, " when it was shown quite conclusively that the incidence of the disease was independent of the food supply, in the sense that one batch of cattle might be *recovering* from the disease at the very time, and in the very paddock, that other members of the same herd were *sickening* of it."

Theiler for a time believed that lamsiekte was caused by a poisonous plant. However, the results of his experiments were entirely negative. Nevertheless he remained so convinced that some form of intoxication best explained the known facts, that he advanced what he termed the "Accumulative Vegetable Poison Theory." This read: "Lamsiekte is *primarily* a disease of the muscular system, caused by a toxin which accumulates in the muscles and is obtained from grasses in certain regions where it is produced under the influence of certain climatical and tellurical conditions." (Theiler, 1912.)

Hence he believed, as did Hutcheon, that, apart from any rôle which might be played by a "want of phosphates," the appearance of lamsiekte was dependent upon "an exciting cause," which he took to be an accumulative plant poison.

It was not until 1918 that Theiler, in the course of personal investigations on the lamsiekte farm Armoedsvlakte, realized the full significance of the depraved appetite of cattle in lamsiekte areas. He observed cattle eating skeletal carcase debris and contracting lamsiekte, and he then succeeded in producing artificial cases of the disease by forcibly administering such debris by the mouth.

The idea of a toxicogenic saprophyte at once suggested itself. The "exciting cause" of lamsiekte, which for so many years had obscured the significance of the mineral deficiency in pastures on lamsiekte and styfsiekte veld, had been found.

From the lamsiekte point of view, there remained only the elaboration of the proof and the investigation of possible preventive methods. Styfsiekte was subsequently proved to be simple aphosphorosis, without any other primary or direct cause.

The sequence of etiological factors in lamsiekte was clearly described by Theiler and co-workers in 1920. It may briefly be repeated here as follows:—

(1) *Deficiency of phosphorus in the soil* leads to
(2) *deficiency of phosphorus in the pasture*; which causes
(3) *deficiency of phosphorus in the animal*; and this produces
(4) *abnormal craving* for bones and carcase debris, which may be
(5) infected with *Clostridium parabotulinum bovis*, which produces
(6) a *toxin*, which causes
(7) *lamsiekte*.

It was shown that this chain of events could be broken in several ways, the simplest and most economical of which was the supplementing of veld pasture with bonemeal, thus supplying the deficient phosphorus and preventing the ingestion of toxic carcase material.

The systematic feeding of bonemeal to cattle in areas where lamsiekte and styfsiekte occurred, and where formerly cattle farming was, at best, a very precarious method of obtaining a living, has made this type of farming not only possible, but very profitable.

In the course of the lamsiekte investigations, described above, it was proved beyond doubt that large areas of South Africa are markedly deficient in phosphates.

The attention of Theiler and co-workers now became centred on the general problem of mineral deficiency in South African pastures, and its effect on live stock, from the nutritional aspect.

Early in 1920, extensive feeding experiments were started on the farm Armoedsvlakte, in the Vryburg district of Bechuanaland, where the previous lamsiekte experiments were carried out.

The results of these experiments, together with the results of soil and pasture analysis, are fully described by Theiler, Green, and Du Toit (1924).

As the present investigations, of which this article is to be regarded as a progress report, are the direct results of the experiments quoted above, a summary of these experiments is here warranted. It was conclusively shown:—

1. That in the areas where the investigations were carried out, phosphorus is the limiting factor in—
   - (*a*) the growth of young stock;
   - (*b*) the condition in older stock;
   - (*c*) the milk yield in cows.

2. That the rate of increase in weight of young growing stock is approximately twice as fast in bonemeal-fed calves as in control calves. Starting with nondescript calves of about 300 lb. live weight, and from 9 to 18 months old, the controls gained on the average 170 lb. in one year. The bonemeal-fed calves gained during the same period and on the same veld 340 lb. in live weight. With this greater increase in live weight is associated earlier maturity and superior quality beef.

3. Similar experiments conducted on young oxen and on mixed herds, gave similar though not quite as striking results.

4. It was shown that the amount of phosphorus required to prevent osteophagia is less than the amount required for optimum growth. Three calves were selected on the grounds of uniformity in age, size, and appearance. The following table gives the results of the experiment in which the first calf received no bonemeal at all, the second just sufficient to prevent osteophagia, and the third calf an excess of bonemeal.

	Calf Number 587.	Calf Number 559.	Calf Number 560.
	lb.	lb.	lb.
Weight, January, 1922	224	237	226
Weight, May, 1923	390	532	578
Increase in weight	166	295	352
Total bonemeal supplied	Nil.	15	75

5. A graph was given to illustrate the variation in amounts of bonemeal required by different classes of stock. Lactating cows (with calf at foot) were shown to require most bonemeal; cows and heifers in calf somewhat less; growing stock up to three years old still less; while of all classes of stock adult oxen and dry non-breeding cows require least.

6. A further experiment showed that bonemeal feeding to lactating cows not only resulted in an average greater milk yield per lactation period, but also in the milk yield being kept up longer and at a higher level of production than in the control animals. The effect on the sucking calf was obvious.

7. It was shown that cattle on a phosphorus deficient diet do not utilize their food economically. Even if they eat a lot, they waste it, and do not give a profitable return in live weight for it. If the deficiency is rectified, by bonemeal feeding, cattle eat more, make much better use of their food, and give much better value in beef for what they eat.

8. A table was given showing the composition (based on dry matter in the plant) of mixed Armoedsvlakte veld grasses at different times of the year. This table showed that the P_2O_5 content of the pasture is highest in spring, shortly after the first rains have fallen and the veld is in active growth. Thus in November, 1919, the P_2O_5 content averaged 0.60 per cent. In December of the same year it had already fallen to 0.32 per cent., and

from then onwards the fall was continuous till mid-winter (June, 1920), when it reached the low level of 0.09 per cent. Broadly speaking, the grazing may be said to be below the normal requirements of mixed stock all the year round.

9. Soil analyses showed that the available P_2O_5 in the soils of the lamsiekte and styfsiekte areas under investigation was as low as 0.001 to 0.0005 per cent.

It is worthy of note that all the feeding experiments were conducted on the nondescript native stock found in the areas under observation.

III. THE PROBLEM TO BE INVESTIGATED.

A study of the literature shows that mineral deficiency of the soil and pasture is of common occurrence in many parts of the world. It is unnecessary for the purpose of this paper to quote extensively from the literature, but mention must be made of the very complete and exceedingly useful review by J. B. Orr (1929).

According to the figures quoted by Orr there is probably no country so deficient in phosphorus as Bechuanaland, where our experiments were carried out. Both the pasture and the soil analysis on Armoedsvlakte have yielded figures for phosphorus which are lower than those obtained from any other part of the world.

Interesting figures are quoted by Orr to show that a mineral deficiency in the soil and pasture is the more likely to lead to a deficiency disease in the animal, the faster growing the animal is. "The faster the growth, the richer must the diet be in all food constituents including inorganic salts, which are needed as constructive material for the formation of new tissue." To prove this it is shown, on the one hand, that the milk of the different species of animals contains more mineral matter, the faster the rate of growth of the young of that species. The following table, taken from Pröscher and Abderhalden in modified form, illustrates this point:—

Species.	Time in Days to Double Weight after Birth.	The Milk of Species contains.	
		Ash.	Ash in grams. per 1,000 cal.
Man	180	0·25	3·7
Cow	47	0·72	10·5
Pig	14	1·03	10·9
Rabbit	6	2·50	15·0

On the other hand, it is shown that fast growing, improved breeds of cattle need more minerals and are therefore more likely to show the effects of a deficiency in the pasture than slow growing native breeds. Orr's remarks on this point—which really forms the pivot of our present investigations—are of such interest that they may be quoted in full:—

> "The rate of growth of cattle in the natural state is slow compared with modern standards, and the amount of milk produced by cows is limited

> to that required by the calf. The improvement of cattle by selective breeding has produced types with a greater rate of growth and a greater capacity for the production of milk. Modern cattle grow at double the rate of the type from which they originated and they reach maturity in about half the time. The domestic cow, under good conditions of feeding, has her first calf when about three years old. 'Native' cattle breed much later. Thus in Nigeria cows do not have their first calf until they are about six years old (Du Toit). Milk production has also increased. In the natural state cows do not produce more than 200 to 300 gallons per lactation period, whereas, in modern dairy breeds, the yield is commonly from 700 to 1,000 gallons. This improvement of the breed has been associated with an improvement in the pastures. Cultivated pastures are much richer in minerals than those grown on natural unimproved soils. Without the improvement of the pastures, improvement of the breed would have been impossible.
>
> "When animals of an improved type, bred on a mineral rich pasture, are transferred to a district with poor pastures, the low mineral content of the poor pastures is liable to be insufficient to support the rate of growth or the capacity of milk production, and consequently mal-nutrition, due to deficiency of minerals, is likely to occur. As a matter of fact, in new countries diseases associated with deficiency of minerals have occurred under these conditions. It is commonly found that when high grade bulls are imported to grade up native cattle, the mortality increases with grading up, i.e. as the rate of growth of the cattle increases. This has occurred in many parts of the British Empire where attempts have been made to improve the breed without ensuring that the feed was sufficient to maintain an improved breed."

In these paragraphs Orr describes exactly what has occurred in many parts of South Africa. On the mineral deficient areas slow growing "scrub" cattle exist, but cannot be said to thrive. If improved breeds are brought into such areas they soon show signs of deficiency and will gradually deteriorate until their progeny is little better than the original scrub animals.

The question then arises: *Can improved breeds of cattle be raised on this deficient veld, provided the missing phosphorus is supplied to the animals?*

A preliminary answer to this question will be found in the following pages.

Indigenous Breeds.

Several types of indigenous cattle have been known in South Africa, such as the Bechuana, Mashona, Damara and Zulu types.

Of the past histories of these types very little is known. Due to the introduction of imported breeds, these native types have been so changed in external conformation and general characteristics, that it is now most difficult, if not impossible, to find typical specimens.

Only one indigenous breed, the present day "*Africander*" breed, has escaped the influence of imported blood, and has been bred pure since the days of the first colonists in the 17th century. This has not been due so much to the outstanding qualities of the original "Africander" stock, which the early settlers found in the possession of the Hottentots, as to the fact that this was the only breed of cattle to be had in those days. Importation of superior types from overseas was quite impossible.

When ultimately European types were imported (the first European cattle to be imported into this country were Fries cattle in the year 1780), the Africander breed had already established itself in the esteem of very many farmers, on account of the wonderful draft powers of the oxen. Throughout the history of this country, the breed has been renowned for its trek oxen.

The most reliable information about indigenous stock we find in the history of the Africander breed, the original home of which may be considered to have been the south-western part of the Cape.

The outstanding characteristics of the breed on the open range may be summarized as follows:—

1. A rather light breed in body weight.
2. Particularly fine and small of bone.
3. Rather leggy.
4. Rather narrow and undeep of body and belly tucked up.
5. Definitely late in sexual and bodily maturity.
6. Although of high fecundity in the sense that cows breed up to a great age, they are notorious as irregular breeders, often missing a breeding season. In many parts of this country, Africander cows, under veld conditions, will calve only once in two years.
7. The cows are very mediocre milkers, giving but little milk over and above the amount needed by the calves. They are, however, good mothers.
8. From the beef point of view, the Africander, if it has not been used as a trek ox, gives fair quality beef, dresses well, but gives a light carcase. As already stated it is a late maturing animal, and as a rule not fit for slaughter until four or five years old.
9. The breed is noted for—
    - (*a*) its powers of endurance as "trek" animals;
    - (*b*) its ability to subsist on poor veld in periods of drought;
    - (*c*) its resistance to tick-borne diseases;
    - (*d*) its ability to withstand adverse climatic conditions.

The bodily conformation described above was looked upon with favour by the farmers, because fine bone, a tendency to legginess, a rather narrow body, and not too big a belly, were associated by them with the physiological ideal build of a trek ox. In fact, old Africander breeders pay considerable attention to these points in the selection of breeding stock.

This same rangy build of the old Africander is found in the nondescript and unimproved stock in many parts of this country. It is especially noticeable in the unimproved native cattle of Bechuanaland.

In the days when cattle were mainly used as draft animals, and the beef and milk sides of the cattle industry were as yet of little economical importance, the conformation described may have satisfied cattle farmers.

With the development, however, of the "ranch dairy" and beef industries, an endeavour was made to improve the body build of our native cattle in conformity with functional requirements. This was done mainly by the introduction of sires of imported breeds and by selection, but, although this systematic breeding has been in progress now for well over a century, the natural tendency to ranginess still remains.

Although the greatest care is being exercised by owners of pure bred and high grade herds in the selection of their bulls and breeding stock, on the basis of good bone, width, and depth of body, moderate length of legs, etc., it has been found necessary continually to introduce new blood and new sires of the desired bodily conformation, to prevent the cattle from developing into "off types."

Not only is the body build liable to become "off type," but experience has taught us that in the improvement of our native stock, the higher grades in many parts of the country lose their natural vitality and resistance to external or environmental conditions, and become less productive. They do not do well and have a reputation of being "soft."

To reinstil constitutional vigour, it has been a common practice amongst ranchers to cross back on to the Africander after a few top-crosses of the native stock with bulls of imported beef breeds.

Such a system of breeding is undesirable, both from the point of view of general breeding principles and from an economical aspect.

In the light of our experience obtained during the lamsiekte and styfsiekte investigations, and as a result of the subsequent feeding experiments, we feel justified in interpreting the phenomena, described above, partially at any rate, in terms of a mineral deficiency in the pastures.

When animals of an improved type, bred on a mineral rich pasture, are imported to grade up native cattle, the low mineral content of the poor pastures is liable to be insufficient to support the *increased* rate of growth and capacity for milk production in the grades, and consequently malnutrition, due to mineral deficiency, is likely to occur. Following in the wake of this malnutrition, we have loss of constitutional vigour, increased mortality and, as the result of an effort on the part of the functionally improved animals to adapt themselves to the deficient nutritional conditions, a change in skeletal development and body conformation.

Whereas in the past all improvement of native stock, under range conditions, was based solely on the application of breeding principles, without any attention to possible mineral deficiencies in the pasture, due regard was paid in the present investigations to the mineral content of the pastures.

IV. OBJECTS OF THE PRESENT INVESTIGATION.

A. *The Main Object* of the experiments was to study, on a phosphorus deficient farm, the influence of a bonemeal ration on the grading up of native cattle with pure bred bulls.

In this connection particular attention was devoted to the influence of phosphorus on:—

1. Fertility.
2. Sexual and body maturity.
3. Retention of constitutional vigour.
4. Grades of indigenous as compared with grades of imported breeds.
5. The various functions of the different breeds (e.g. milk production, beef production, dual purpose function, draft function).

B. *The Second Object* of the experiment was to study the dietetic side of the problem, special attention being devoted to the influence of bonemeal feeding on:—

1. Weight of calves at birth.
2. Growth of calves.
3. Increase in body weight.
4. Retention of condition during winter.
5. Quality and quantity of beef.
6. Quality and quantity of milk.
7. Skeletal development.
8. Craving for bones (pica or osteophagia).

C. *The Third Object* of the investigation was a purely veterinary one, namely, to study the influence of bonemeal on the incidence of disease.

It has, of course, been known that the incidence of lamsiekte and styfsiekte would decrease greatly if bonemeal was fed; but the present object was to observe whether animals receiving bonemeal would show a greater resistance to diseases in general than the control animals receiving no bonemeal.

Briefly the object of the investigation may thus be said to comprise:—

A. *The Breeding Problem,*
B. *The Nutritional Problem.*
C. *The Problem of Health and Disease,*

on a phosphorus deficient pasture.

V. PLAN OF THE EXPERIMENT.

The experiment was started on the 5th of March, 1925, on the Lamsiekte Experiment farm "Armoedsvlakte," in the district of Vryburg in Bechuanaland (see Figure 1).

A. THE EXPERIMENTAL ANIMALS.

(*a*) Selection of the Basis Herd Cows.

As the foundation stock of the experiment 200 cows were collected, some locally and the others from cattle on the various outstations of this Division. No attempt was made at selection, and as a result, the Basis Herd represented a very mixed collection of cows, very typical of the nondescript mob so often met with on cattle farms in the ranching areas (see Figures 2 to 9). A fair percentage of the animals showed the presence, somewhere in their ancestry, of Africander blood; a few could, with a fair stretch of imagination, be called Fries and Shorthorn types, but by far the greatest number could not be called anything but "native" stock, varying in size and conformation from the small-bodied, pot-bellied and large-horned Mashona type, to the leggy, narrow, rangy type already described.

In colour blacks and reds predominated with sprinklings of yellow, red and white, black and white, and brindle.

As to age, the herd consisted of two-year-old heifers to ten-year-old cows.

With the exception of, say, 10 to 15 cows the milk production was not higher than 1 gallon a day, and the lactation period scarcely lasted till the weaning age of the calves at nine months.

(*b*) The Four Herds, their Selection and Function.

To make the experiment as complete as possible, it was decided to grade up the Basis Herd cows in four different directions, according to function The 200 cows were therefore divided into four groups of 50 each:—

Group I: to be graded up with Africander bulls.
Group II: with Fries bulls.
Group III: with Red Poll bulls.
Group IV: with Sussex bulls.

In this way the trek, milk, dual purpose and beef breeds of cattle would all be represented.

The Africander was selected, not only as representing the "trek type" par excellence in this country, but specially too because it was the one breed indigenous to South Africa which has been kept pure. As it was part of the experimental programme to make a comparative study of the effect of bonemeal feeding on grades of indigenous and imported breeds, the inclusion of the Africander was essential.

The Fries was chosen as the dairy breed proper, because, of the various dairy breeds in this country, it is by far the most popular and most widely distributed.

Amongst the dual purpose breeds the Red Poll was selected for no special reason, apart from the fact that at that time the breed had but recently been introduced into the country and it was thought that the experiment would provide some valuable data as to the suitability of the breed in "ranch dairy" areas.

For a representative of the beef type proper the Sussex was chosen because the breed had already proved itself to be hardy veld animals, which would do well under South African conditions.

The division of the Basis Herd cows into the four herds was obviously very difficult because of their nondescript character, but, as far as possible, the cattle for the four herds were selected according to any tendency in their external conformation towards any of the types named above.

(*c*) Bonemeal and Control Groups.

After a sufficiently long preliminary period of bonemeal feeding to all the cows, each herd of 50 cows (i.e. the Africander, Fries, Red Poll and Sussex herds) was again sub-divided into a Bonemeal and a Control group, the former in each herd numbering 34 animals and the latter 16 animals (i.e. in the approximate proportion of two Bonemeal to one Control). The experiment may thus be said to have been divided, at the start, into two parallel experiments, the bonemeal experiment and the control experiment, both conducted on identical lines in every respect, except only with regard to the bonemeal feeding.

(*d*) Breeding Methods.

It was decided to grade up the four herds with pure-bred bulls for at least three generations (i.e. up to $\frac{7}{8}$ pure), and, if possible, for four generations, or up to 15/16 pure bred. This would mean that the experiment would continue for at least 15 to 17 years and it was thought that that was a sufficiently long period in which the objects of the experiment could be investigated.

(*e*) The Herd Bulls, their Selection, Type, and Breeding.

Two bulls were obtained for each herd, but, owing to the fact that the cost of the experiment had to be curtailed, it was impossible to get the best types of these breeds. Hence the first bulls were rather on the poor side, the Fries definitely lacking the necessary robust constitution so essential in sires in areas of semi-arid pastures and climatic extremes, such as are found in Bechuanaland. The Red Poll bulls, too, were rather too rangy of body build. The Sussex bulls conformed more to the desired type and the Africanders, although of good constitution, were by no means typical of the breed. These last bulls were not registered animals. These bulls sired the first generation of calves (i.e. three successive batches in 1926, 1927, and 1928), and were disposed of directly the third service season had been completed.

Since then two new bulls of each breed have been purchased to sire the second generation of calves, and particular care was taken to select animals of outstanding constitution, good bone, close to the ground, good depth and width of body, good capacity and quality.

With regard to the Fries and Red Poll bulls, care was taken not to select animals with too high milk records, the reason for this being that the experiment was designed as a " veld " experiment, with no additional feeding whatever, except in the case of the bulls. Under such a system of " ranch dairying," very high milk records would be wasted, as the cows could not obtain sufficient food from the pasture to allow of the full development of a very high milk production. Care was further taken, that the new bulls of each breed should not be related to each other, and should not show marked evidence to close breeding or inbreeding in their pedigrees. This was done in order to obviate complications in the breeding system which was to be applied in the experiment.

(*f*) SERVICE AND CALVING SEASONS.

As will be pointed out later, the rainy season in Bechuanaland is but a short one, extending approximately from the middle of December to the end of March. Hence the period when the pasture is green and the cows are most likely to take the bull, is limited to these months.

However, in order to allow the calves to be dropped subsequent to the first rains following the long and dry winter (May to December), when the grass is once more green and nourishing, the service season was extended from the 20th of February to the 20th May of each year. This allows the calving season to fall well within the months of the best pasture conditions, thus ensuring a good milk supply for the calves.

In the case of the first generation of calves (1926, 1927, and 1928), the bulls were run with the cows for the duration of the service season, no control being kept, as to which of the two bulls served a cow, or how many times each cow was served. Consequently, after three seasons the first bulls had to be discarded, in order to prevent the batch I heifers of the first half-bred generation from being served by their own sires.

This system has since been changed with the introduction of the new bulls. Service now takes place in the kraals every morning after bonemeal feeding, the two bulls being put to successive batches of heifers. The system may be illustrated as follows: During the service season of 1928 only the batch I (1926) heifers were old enough to breed from. They were all served by Bull No. I. In 1929 batch II (1927) heifers as well as batch I (1926) heifers will be bred from. Batch I will now be served by Bull No. II and batch II by Bull No. I. Continuing in this way, it will be seen that the present bulls will be able to sire three batches of $\frac{3}{4}$-bred calves from each of the three batches of $\frac{1}{2}$-bred cows, i.e. over a period of five years.

Careful records are kept, not only of the sire of each calf, but also of the dates upon which the cows are served. If cows keep coming back to the bull, or if they do not come on heat at all, they are examined to determine the cause. This supplies valuable data with regard to factors affecting fertility, sterility, sexual maturity, etc. Furthermore, the bulls are allowed to serve a cow only twice on any one date. This is done to conserve the animals' vitality and breeding powers, and to prevent the bulls from spending themselves on one cow, as is the common rule when bulls are allowed to run with the cows. It is nothing unusual under a system of veld service for a bull to serve one cow 10 to 12 times during the course of a day.

(*g*) BREEDING AGE, NUMBER OF CALVES PER COW, WEANING AGE.

In order to keep the number of cattle in the experiment within the limits of the carrying capacity of the available veld, and particularly to reduce the number of batches of calves, the basis herd cows were allowed to give three crops of calves (i.e. in 1926, 1927, and 1928), and were discharged from the experiment in November, 1928, after the last calves had been weaned. The three half-bred batches will again each give three batches of calves so that in all there will be nine $\frac{3}{4}$-bred batches and 27 batches of $\frac{7}{8}$-bred grades. It is obvious that where accurate records are essential, the number of batches must be kept down if the experiment is not to become too unwieldly.

Heifers of all four breeds are put to the bulls when two years to two years and three months old. In the case of the European breeds this has proved to be quite satisfactory, but in the Africander herd the service figures, as will be shown later, are not quite so good. However, the experimental programme will not allow for differentiation in this respect. Calves are weaned in November when 9 to 11 months old.

(*h*) CULLING AND SELECTION OF BREEDING STOCK.

Amongst the controls no culling or selection of breeding stock takes place, for the simple reason that lamsiekte, styfsiekte, and poverty have reduced the numbers to such an extent, since the start of the experiment, that the control herd is not likely to survive the half-bred stage.

Up to the present, selection of desirable types and culling of undesirable animals have been impracticable in the bonemeal groups of the first generation, because of the small numbers of heifers in each batch.

Within the next few years, however, the number of second generation heifers ought to be sufficient to allow of extensive selection and culling, and this practice will then be continued throughout the duration of the investigation.

(*i*) CASTRATION, NUMBERING, BRANDING, AND CULLING.

In the past bull calves were castrated at the age of approximately six months. In 1927, however, it was found that several batch I half-bred heifers amongst the European breeds were in calf to these young bulls and hence, since then, bull calves have been castrated at two months of age, with the Burdizzo pincers. The results have proved entirely satisfactory.

Calving, of course, takes place in the open, no special provision being made to place heavily pregnant cows in a separate camp. Every morning during the calving season when the cattle come in for dosing, the calves born over-night are registered against their dams and numbered with an eartag. At weaning age each calf's eartag is checked against the number of the mother and against the registered number in the calf book, and, if found correct, the calf is permanently branded on the off thigh. This being done, the calf is transferred from the calf book to the herd register. Naturally, all the bulls and basis herd cows are also numbered and branded.

At weaning time culling of the young stock takes place. The tollies are of no further use in the breeding experiment, and are passed into the slaughter test experiment or discharged.

(*j*) BONEMEAL FEEDING.

All cattle are brought in from the camps to the homestead daily for counting and inspection. All the cattle too are passed through the bonemeal dosing crushes, but the controls which are marked with a band of blue paint round the horns (or by a patch of blue paint on the poll in the case of polled cattle) are passed through without being dosed.

The bulls, cows, heifers in calf, and lactating cows receive 5 ozs. of bonemeal daily (except Sundays), whilst all growing and dry stock receive a daily allowance of 3 ozs. The method of dosing is very simple and effective. Leading out of a large collecting kraal are two 66 feet long wooden crushes running parallel to each other and about 16 feet apart (see Fig. 26). The crushes are four feet high and two feet two inches wide. The cattle are driven into these crushes, and while this is being done, a native stands on the near side of the cattle, thus forcing them to pass their heads to the off side (see Fig. 27). When the crushes are full dosing starts. A native, standing on the right side of the animal to be dosed, catches hold of the left horn with his left hand and slips his right hand between the lips and grips the right cheek of the animal. This is sufficient to control the head, and to make the animal open its mouth. The actual dosing is done by the stockman. He grips the tongue with his left hand, the thumb below and the other four fingers over the tongue, pulls it out and turns it to the left. With his right hand he takes a spoonful (3 oz. to 5 oz.) of moistened bonemeal from a bucket, held by a second native, and places this on the right side of the tongue, just in front of its base. With the back of the spoon he pushes the mass of bonemeal over the base and directly lets go the tongue (see Fig. 28). The animal cannot now get rid of the bonemeal and is sure to get its full dose.

While the second crush-full is being dosed, the first crush is being emptied and refilled, so that at no time need the dosing be interrupted.

Although from the description, dosing may appear to be a complicated and difficult procedure, in reality it is very simple indeed. At Armoedsvlakte four natives and one European assistant dose from 200 to 250 head in an hour, with ease.

Cattle soon become used to being dosed, and it often happens that more than one will open its mouth and partially protrude its tongue as soon as it is approached.

Calves are not dosed until weaned at 9 months of age. Perhaps the most difficult part of bonemeal feeding is to get these youngsters used to dosing. Not only will they frequently lie down and refuse to pick up their heads or try to jump out of the crush, but they will retract the tongue so that the operator has great difficulty in getting hold of it. However, usually after three weeks' training, they have become accustomed to dosing.

As already mentioned, the bonemeal is fed moist. This facilitates the filling of the spoon, allows the assistant to gauge the correct dose more accurately than with dry bonemeal, makes the actual dosing simpler, and prevents the animals from blowing the meal out of their mouths or drawing fine particles into their lungs.

With bonemeal at £8. 10s. per ton, the cost of bonemeal feeding works out at 4s. 11¾d. per animal per year, when the ration is 3 oz. per day (except Sundays) and 8s. 3¾d. when the ration is 5 oz.; or in round figures: 5s. and 8s. 4d. per animal per year, respectively.

(*k*) Weighing of Experimental Animals.

All cattle are weighed every two months. Since exercise and drinking have quite an appreciable effect upon body weight, the cattle, after being driven to the homestead, are first dosed, then watered, and finally weighed.

Calves are weighed at birth, and from then onwards, on the same days as the other stock.

(*l*) Osteophagia or " Pica " Testing.

Although the amounts of bonemeal fed to the different classes of stock, are considered sufficient for optimum growth and health, osteophagia (" pica " or craving for bones) does occur even in the bonemeal-fed cattle, especially in pregnant and lactating cows during the dry seasons of the year.

In order to have a record of the degree of osteophagia, all cattle over nine months of age are " tested " every 14 days.

The method is as follows:—

From a big collecting kraal, batches of six to eight animals are passed into a small kraal, in which there are troughs containing sterilized rotten bones. Any animals which pick up and chew one of these bones are marked down as " rotten bone cravers " or " bad cravers," and are passed into another collecting kraal. The cattle which do not touch the rotten bones are passed into a second small kraal, containing sterilized bleached bones or " sweet bones " in troughs. Any animals picking up and chewing these are marked down as " sweet bone cravers " or " mild cravers." The animals which touch neither rotten nor sweet bones are recorded as " non-cravers."

(*m*) Milking and Butterfat Testing.

No records were kept of the milk production nor were butterfat tests conducted on the basis herd cows. These constituted such a mixed mob of cattle that, apart from determining the approximate average daily yield for the herd, it was not thought worth while to take records.

Now, however, that the first batch of half-bred heifers are calving, the individual milk yields and butterfat percentages are to be recorded in the case of the Fries and Red Poll herds.

The method adopted for the recording of the milk yields is not perhaps an ideal or very accurate one, but it is the most practical under conditions obtaining on a South African ranch, where the cows are never stabled and are totally veld fed.

For the first three months after the birth of the calf the cows are milked twice daily, in the morning and in the evening. In the morning, the calves are allowed to suck and strip one half of the udder, while the other half is milked for recording purposes. After weighing, the milk is given back to the calf, if necessary. In the afternoon the process is reversed, the calf being allowed to suck the half which had been milked during the morning, while the remaining half is again milked and the milk weighed for recording purposes.

Every seventh day the calves are not allowed to suck at all. The whole udder is milked, morning and evening, for recording purposes and the weighed milk is fed to the calf from a bucket. This is done in order to get some idea of the experimental error due to the system of milking.

From three months old till weaning age at nine months, the calves are allowed to suck in the morning only, one day one-half of the udder being given to the calf, while the other half is milked and the milk weighed. The next day the halves are reversed. On the seventh day the whole udder is milked and the weight recorded. In each case the milk is given back to the calf, if necessary.

Butterfat tests of each cow are made on every 7th day and on a composite sample of the morning's and evening's milk. The Gerber method is employed for this determination.

The amount of " solids not fat " is also determined by means of Fleischman's formula, viz.: Total solids $= \frac{1}{4}\,G + 1.2\,F + 0.14$ where G is the specific gravity (taken with a Quevenne Lactometer) and F the percentage butterfat as determined above.

(*n*) Dipping, Clinic, etc.

Although the area is comparatively free of tickborne diseases, the veld is heavily infested with the " bontlegged " tick (*Hyalomma aegyptium*) and the blue tick (*Boophilus decoloratus*). The first named is especially troublesome. In summer they attack cattle, particularly under the tail, between the hind legs, in the axillae and in the tail switch, and produce small deep-seated abscesses of a virulent and chronic nature.

To combat this affection, all cattle are dipped weekly in an arsenical solution, from September until the end of April.

The ordinary horse-fly (*Hippobosca* spp.) also constitutes a veritable pest during the summer months, and it has been found that by adding a gallon of whale oil to the dip, at every dipping, these flies are less troublesome to the cattle.

As the cattle are passed through the crushes every morning for dosing, they are carefully examined for wounds, tick abscesses, ophthalmia, ringworm, etc., and if necessary treated. The bulls are temperatured every day.

In the past no records were kept of the clinical cases, but since the study of the influence of bonemeal feeding upon the incidence of disease is included in the experimental programme, all cases are now carefully recorded.

B. THE FARMS.

The Lamsiekte Experiment Station consists of the two farms, " Armoedsvlakte " and " Biesjiesvlakte," which together cover an area of 7,000 morgen (14,777 acres). Armoedsvlakte is situated some five miles west of Vryburg, in a region of dolomite outcrops, while Biesjiesvlakte lies some 12½ miles west again of the first farm, in a sandveld area.

1. " *Armoedsvlakte.*"—This farm has very shallow soils, varying from three to 24 inches in depth, with an average depth of not more than nine inches. Only 20 out of the 7,811 acres are arable.

The soil surface is broken at short intervals by low, linear outcrops of dolomite, rising a few feet out of the ground and varying from two to 60 yards in width.

Along these outcrops bushes are plentiful, especially " vaalbos " (*Tarchonanthus camphoratus*), which is evergreen in nature and provides excellent shelter, both against the summer heat and the cold winter winds. In between the outcrops, the grass veld is dotted all over with " rosyntjie bos " (*Grewia cana*). These bushes are deciduous perennials, which, due to wind erosion, grow on little hillocks, some eight to 18 inches higher than the surrounding veld. Both the vaalbos and rosyntjie-bos are said to be eaten by cattle, but this has never been observed on Armoedsvlakte.

The veld consists of " sweet " grasses with the exception of such genera as *Themeda*, *Heteropogon*, *Andropogon*, *Cymbopogon*, etc. The grass colonization is poor; the individual grass tufts are of good size and firmly rooted, and a fair percentage of the grasses are bi- or perennials.

In spring, after the first rains, the veld is not quite as quick in shooting as on Biesjiesvlakte, but it is very resistant to drought, and to tramping out by stock. This last fact is evidenced by the observation that, although the farm has been heavily stocked for the last 15 years, no signs of veld deterioration are as yet visible.

To summarize, Armoedsvlakte may be called a " late, warm, hard-veld farm."

2. " *Biesjiesvlakte.*"—This farm is 3,300 morgen (6,966 acres) in extent and differs greatly from Armoedsvlakte in very many respects.

It is situated in soft sand country; has moderately deep soils, with only occasional " Jonas klip " outcrops on the higher parts of the farm. A " vlei " runs right through the farm, and, dotted over the veld, are a number of small " pans " which, in olden times, were practically full of water for the greater part of the year, and afforded good duck shooting; but which nowadays, seldom hold water for more than a few days during the rainy season.

The farm is, with the exception of a few " rosyntjiebos," quite devoid of bush and appears bare and windswept, affording no protection whatever to stock.

The veld is sweet, more so than on Armoedsvlakte, but the grass colonization is more sparse and the individual tufts are smaller and but poorly rooted. The majority of the grasses are annuals.

The veld is very responsive to rains in spring, shooting up several inches per week, but it is very susceptible to drought.

As may be expected from the nature of the soil, the veld is easily tramped out by stock, and great care has to be exercised continually in this respect, to prevent permanent veld deterioration.

Compared with Armoedsvlakte, Biesjiesvlakte may be said to be an " early, cold, soft-veld farm."

(*a*) Stock Camps, Bull Camps, Paddocks, etc.

Both farms are fully fenced.

Armoedsvlakte is divided into seven stock camps, varying in size from 200 to 800 morgen. Besides these there are five bull camps, four paddocks, and a number of experimental grass and grazing plots.

Although the outlying camps are at least three miles from the homestead, the cattle are brought in every day for inspection and bonemeal dosing. This is possible, because the veld is tenacious and not easily tramped out.

Biesjiesvlakte is divided into four, approximately equal, camps of about 800 morgen each. Due, however, to the " soft " nature of the veld, it has been found necessary to erect crushes in the outlying camps, and dose the cattle away from the homestead, in order to prevent veld deterioration.

The camps on Biesjiesvlakte are without doubt too large, but the question of how big or how small camps should be, in order to obtain maximum results from grazing by cattle, is one of such importance that it warrants fuller discussion. The problem of " good range management " is without doubt of quite as much importance to successful ranching as " good animal management," and hence it is proposed to discuss this question in some detail elsewhere.

(*b*) Rotation of Grazing.

Because of the widely different nature of the pasture on the two farms, it has been the practice, up to the end of 1928, to interchange the cattle on the two farms, every two months, in order to equalize grazing conditions as much as possible. This, however, has perforce been stopped, because the Fries and Red Poll cows of the first generation are now being submitted to milk recording. The necessary facilities for this procedure (milk sheds, calf paddocks, laboratory equipment, labour, etc.) are only available at Armoedsvlakte, and hence the Fries and Red Poll herds will remain there, while the Africander and Sussex herds will be kept at Biesjiesvlakte.

On both farms too the cattle are moved from one camp to another according to the season of the year, the condition of the pasture, the presence or absence of poisonous plants, etc.

Experience has shown that certain camps are good summer camps, and that others are better winter camps. Thus, e.g., the better sheltered camps are used for the cows and calves in winter. At certain times each year one or two camps become dangerous to stock, because of poisonous plants. The worst of these is " slangkop " (*Urginea* spp.), but although the farms have been cleared of this plant, other as yet unknown plant poisons have been responsible for deaths amongst the stock in a few of the camps during definite periods of the year. Hence, during these periods the cattle are removed to " healthy " camps.

The question of systematic camp rotation also forms part of the general problem of good range management, and will be discussed more fully elsewhere.

(*c*) WATER SUPPLY.

That for general health a plentiful and clean water supply should always be within easy reach of the cattle, is a well established principle in animal management. Under climatic and pastoral conditions, such as those prevalent in Bechuanaland, where shade temperatures go up to 105° F. and over, and where the pasture is dry for at least seven months in the year, a good and plentiful water supply is of vital importance.

As no surface water is available, the necessary supplies are obtained from boreholes, either by the aid of windmills (see Fig. 29) or small oil engines.

However, there are times, especially in early summer, when there is little or no wind. Hence allowance must be made for sufficient water storage.

On both farms all the camps are supplied with reservoirs of a minimum capacity of 10,000 gallons, and spacious concrete or stone drinking troughs (Fig. 29).

(*d*) KRAALS, CRUSHES, DIPS, STABLES, ETC.

On each farm, near the homestead, there are large wooden kraals, subdivided for sorting purposes into a sufficient number of smaller kraals. As part of the kraal system, there are the double dosing crushes, leading from one big collection kraal to a second collection kraal; the cattle dip also forms part of the kraals to facilitate the handling of the cattle.

At Armoedsvlakte, new milking sheds, sick boxes and bull boxes are to be erected shortly. In the meantime the milking is done in lean-to stables which were formerly used for experimental purposes.

At Biesjiesvlakte new stables, making provision for bull and sick boxes, have been completed.

A very commodious and fully equipped laboratory was built at Armoedsvlakte, for purposes of lamsiekte research, and is now available for laboratory work in connection with the experiment.

The Station is also supplied with a complete set of meteorological instruments.

C. EXPERIMENTAL RECORDS.

The following experimental records are kept:—

1. *The Herd Book.*—A loose-leaf ledger, giving on the left-hand page full particulars of the breeding stock, and on the

right-hand page full particulars of the progeny of each cow.

2. *The Stock Book.*—In which each animal has a separate page, bearing the number of the animal, on which full particulars are recorded as to date of birth, number of sire, number of dam, dates of preventive inoculations, dates and diagnoses of diseased conditions, etc.
3. *A Bull Register.*—Containing name, experimental number, breed number, and full pedigree of each bull, together with name of breeder, etc.
4. *A Service Register.*—Giving particulars of service dates of each cow, which bull was used, whether served once or twice on any one date, etc.
5. *A Calf Book.*—Giving date of birth, weight at birth, colour and distinguishing marks, sex, number of dam, etc.
6. *A Weight Book.*—Giving the results of the bi-monthly weighings.
7. *A Pica Book.*—Giving the results of the fortnightly pica tests.
8. *A Clinical Case Book.*—In which full particulars are kept of all clinical cases amongst the experimental animals.
9. *A Milk Record Book.*—Giving the daily, weekly, and monthly milk yields of each cow.
10. *A Butterfat Test Register.*—Giving the results of the weekly butterfat tests on the milk of each cow, and also of the tests for " solids not fat."
11. *A Meteorological Register.*—Giving the results of the daily meteorological readings.

Duplicate copies of the data obtained are forwarded monthly to the Experiment Records Office of the Division at Onderstepoort, and here comparative graphs and charts are compiled for each herd, so that the experimental results are always kept up to date for purposes of reference.

VI. EXPERIMENTAL RESULTS.

As already stated, the investigation was begun on the 18th March, 1925. The present Progress Report covers the period from that date up to 30th June, 1928. During this period the Basis Herd Cows have given birth to three batches of first generation calves and the first batch of half-bred heifers have been put to the bull.

It is quite evident that, as yet, little can be said about certain of the items on the experimental programme, but the data so far obtained are sufficient to warrant definite opinions to be expressed with regard to some points, and to allow of tentative opinions to be formulated with regard to others.

A. BREEDING RESULTS.

(*a*) FERTILITY.

(For detailed figures in this connection see table No. 1.)

TABLE No. 1.

		Number of cows in experiment from start, 18/3/25.	Number of cows added to experiment since 18/3/25.	Total number of cows in experiment.	Number of cows still in experiment on 15/6/28.	Percentage of cows still in experiment on 15/6/28.	Number of cows which died in experiment up to 15/6/28.	Percentage of cows which died in experiment up till 15/6/28.	CAUSES OF DEATH.									
									Lamsiekte.	Poverty.	Vegetable poisoning.	Fractures—destroyed.	Quarter-evil.	Tuberculosis.	Arsenical poisoning.	Accidental deaths.	Gangrenous pneumonia.	Unknown causes.
AFRICANDER	Bonemeal	35	4	39	23	59·0	5	12·8	1	—	—	1	—	1	1	—	—	1
	Control	*16*	*7*	*23*	*1*	*4·3*	*19*	*82·6*	*14*	*3*	*1*	—	*1*	—	—	—	—	—
FRIESLAND	Bonemeal	34	8	42	28	66·7	4	9·5	1	—	1	1	—	—	—	—	1	—
	Control	*17*	*8*	*25*	*3*	*12·0*	*16*	*64·0*	*6*	*2*	*2*	*3*	—	—	*2*	—	—	*1*
RED POLL	Bonemeal	37	4	41	25	61·0	3	7·3	1	—	1	—	1	—	—	—	—	—
	Control	*16*	*6*	*22*	*3*	*13·6*	*13*	*59·1*	*9*	—	*2*	*1*	—	—	*1*	—	—	—
SUSSEX	Bonemeal	33	2	35	28	80·0	2	5·7	—	—	1	—	—	—	—	1	—	—
	Control	*16*	*6*	*22*	*6*	*27·3*	*13*	*59·1*	*11*	*1*	*0*	*1*	—	—	—	—	—	—
Bonemeal: Total		139	18	157	104	66·2	14	8·9	3	—	3	2	1	1	1	1	1	1
Control: Total		*65*	*27*	*92*	*13*	*14·1*	*61*	*66·3*	*40*	*6*	*5*	*5*	*1*	—	*3*	—	—	*1*
GRAND TOTAL		204	45	249	117	47·0	75	30·1	43	6	8	7	2	1	4	1	1	2

		Number of cows discharged from experiment up till 15/6/28.	REASONS FOR DISCHARGE.						Number of cows which gave birth to 3 calves in 3 years.	Number of cows which could have given birth to 3 calves in 3 years.	Percentage of cows which gave birth to 3 calves in 3 years.	Number of cows which gave birth to 2 calves in 3 years.	Percentage of cows which gave birth to 2 calves in 3 years.	Number of cows which gave birth to 1 calf in 3 years.	Percentage of cows which gave birth to 1 calf in 3 years.
			Sterility.	Old age.	Styfsiekte.	Contagious abortion.	Other diseases.	Transferred to other experiments.							
AFRICANDER	Bonemeal	11	—	1	—	—	—	10	19	28	67·9	7	25·0	2	7·1
	Control	*3*	—	—	—	—	*1*	*2*	*0*	*3*	*0*	*2*	*66·7*	*1*	*33·3*
FRIESLAND	Bonemeal	10	1	3	1	1	—	4	18	24	75·0	5	20·8	1	4·2
	Control	*6*	*1*	—	—	—	*1*	*4*	*0*	*4*	*0*	*3*	*75·0*	*1*	*25·0*
RED POLL	Bonemeal	13	—	4	—	—	—	9	16	26	61·5	8	30·8	2	7·7
	Control	*6*	—	—	—	*1*	*1*	*4*	*0*	*7*	*0*	*3*	*42·9*	*4*	*57·1*
SUSSEX	Bonemeal	5	1	—	—	—	1	3	19	31	61·3	11	35·5	1	3·2
	Control	*3*	—	—	—	—	—	*3*	*0*	*6*	*0*	*5*	*83·3*	*1*	*16·7*
Bonemeal: Total		39	2	8	1	1	1	26	72	109	66·1	31	28·4	6	5·5
Control: Total		*18*	*1*	—	—	*1*	*3*	*13*	*0*	*20*	*0*	*13*	*65·0*	*7*	*35·0*
GRAND TOTAL		57	3	8	1	1	4	39	72	129	55·8	44	34·1	13	10·1

		Number of cows which gave birth to 2 calves in 2 years.	Number of cows which could have given birth to 2 calves in 2 years.	Percentage of cows which gave birth to 2 calves in 2 years.	Number of cows which gave birth to 1 calf in 2 years.	Percentage of cows which gave birth to 1 calf in 2 years.	Number of cows which gave birth to 1 calf in 1 year.	Number of cows which could have given birth to 1 calf in 1 year.	Percentage of cows which gave birth to 1 calf in 1 year.	Number of half-bred Batch I 1926 calves.	Number of cows which could calve in 1926.	Percentage of cows which calved in 1926.	Number of half-bred Batch II 1927 calves.	Number of cows which could calve in 1927.	Percentage of cows which calved in 1927.	Number of half-bred Batch III 1928 calves.	Number of cows which could calve in 1928.	Percentage of cows which calved in 1928.	Total number of calves 1926, 1927, 1928.	Possible number of calves, 1926, 1927, 1928.	Percentage of cows which calved.
AFRICANDER	Bonemeal	5	5	100	—	—	1	3	33·3	29	35	82·9	32	33	97·0	23	29	79·3	84	97	86·6
	Control	*2*	*9*	*22·2*	*7*	*77·8*	*7*	*11*	*63·6*	*15*	*18*	*83·3*	*6*	*14*	*42·9*	*2*	*6*	*33·3*	*23*	*38*	*60·5*
FRIESLAND	Bonemeal	12	12	100	—	—	3	4	75·0	31	32	96·9	34	37	91·9	27	31	87·1	92	100	92·0
	Control	*2*	*6*	*33·3*	*3*	*50·0*	*8*	*11*	*72·7*	*9*	*13*	*69·2*	*8*	*13*	*61·5*	*5*	*9*	*55·6*	*22*	*35*	*62·8*
RED POLL	Bonemeal	8	10	80·0	2	20·0	5	5	100	29	37	78·4	33	36	91·7	27	30	90·0	89	103	86·4
	Control	*0*	*1*	*0*	*1*	*100*	*4*	*11*	*36·4*	*4*	*15*	*26·7*	*8*	*12*	*66·7*	*3*	*7*	*42·9*	*15*	*34*	*44·1*
SUSSEX	Bonemeal	1	3	33·3	1	33·3	1	1	100	25	34	76·5	31	34	91·2	28	32	87·5	84	100	84·0
	Control	*1*	*5*	*20·0*	*3*	*60·0*	*6*	*10*	*60·0*	*9*	*16*	*56·3*	*7*	*13*	*53·8*	*6*	*9*	*66·7*	*22*	*38*	*57·9*
Bonemeal: Total		26	30	86·7	3	10·0	10	13	76·9	114	138	82·6	130	140	92·9	105	122	86·1	349	400	87·3
Control: Total		*5*	*21*	*23·8*	*14*	*66·7*	*25*	*43*	*58·1*	*37*	*62*	*59·7*	*29*	*52*	*55·8*	*16*	*31*	*51·6*	*82*	*145*	*56·5*
GRAND TOTAL		31	51	60·8	17	33·3	35	56	62·5	150	200	75·0	159	192	82·8	121	153	79·1	431	545	79·1

TABLE No. 2.

			Number of half-bred calves born.	Number of cows which could calve.	Actual number of calves to percentage possible number of calves.	Number of bull calves.	Number of heifer calves.	Percentage of bull calves.	Average weight of bull calves at birth.	Average weight of heifer calves at birth.	Average weight of all calves at birth.	Number of calves which died up till 15/6/28.	Percentage of calves which died up till 15/6/28.	CAUSES OF DEATH.										Number of calves discharged up till 15/6/28.	Percentage of calves discharged up till 15/6/28.	REASONS FOR DISCHARGE.						Number of calves still in experiment on 15/6/28.	Percentage of calves still in experiment on 15/6/28.
														Lamsiekte.	Sweating sickness.	Vegetable poisoning.	Fractures—destroyed.	Quarter-evil.	Traumatic pericarditis.	Arsenical poisoning.	Accidents.	Pneumonia.	Unknown causes.			Transferred to breeding stock.	Culled.	Transferred to controls.	Slaughter test, 1928.	Slaughter test, 1929.	Transferred to other experiments.		
AFRICANDER.	1926	Bonemeal	29	35	82·9	15	14	51·7	—	—	—	1	3·5	—	—	—	—	1	—	—	—	—	—	28	96·5	6	7	4	5	3	3	—	—
		Control	*15*	*18*	*83·3*	*7*	*8*	*46·7*	—	—	—	*6*	*40·0*	*2*	—	*3*	*1*	—	—	—	—	—	—	*9*	*60·0*	*5*	—	—	*2*	*1*	*1*	—	—
		Total	44	53	83·0	22	22	50·0	—	—	—	7	15·9	2	—	3	1	1	—	—	—	—	—	37	84·1	11	7	4	7	4	4	—	—
	1927	Bonemeal	32	33	97·0	12	20	37·5	66·7	65·4	65·6	1	3·1	—	—	—	—	—	—	—	1	—	—	11	34·4	—	11	—	—	—	—	20	62·5
		Control	*6*	*14*	*42·9*	*4*	*2*	*66·7*	*71·0*	*66·5*	*69·5*	*0*	—	—	—	—	—	—	—	—	—	—	—	*4*	*66·7*	—	—	—	—	—	*4*	*2*	*33·3*
		Total	38	47	80·9	16	22	42·1	68·8	65·9	67·5	1	2·7	—	—	—	—	—	—	—	1	—	—	15	39·5	—	11	—	—	—	4	22	57·9
	1928	Bonemeal	23	29	79·3	9	14	39·1	62·1	62·8	62·5	0	—	—	—	—	—	—	—	—	—	—	—	0	—	—	—	—	—	—	—	23	100
		Control	*2*	*6*	*33·3*	*0*	*2*	—	—	*64·0*	*64·0*	*0*	—	—	—	—	—	—	—	—	—	—	—	*0*	—	—	—	—	—	—	—	*2*	*100*
		Total	25	35	71·4	9	16	36·0	62·1	63·4	63·3	0	—	—	—	—	—	—	—	—	—	—	—	0	—	—	—	—	—	—	—	25	100
	Total	Bonemeal	84	97	86·6	36	48	46·5	64·4	64·1	64·1	2	2·4	—	—	—	—	1	—	—	1	—	—	39	46·4	6	18	4	5	3	3	43	51·2
	Total	*Control*	*23*	*38*	*60·5*	*11*	*12*	*47·8*	*71·0*	*65·3*	*66·8*	*6*	*26·1*	*2*	—	*3*	*1*	—	—	—	—	—	—	*13*	*56·5*	*5*	—	—	*2*	*1*	*5*	*4*	*17·4*
		TOTAL	107	135	79·3	47	60	43·9	67·7	65·7	65·4	8	7·5	2	—	3	1	1	—	—	1	—	—	52	48·6	11	18	4	7	4	8	47	43·9
FRIESLAND.	1926	Bonemeal	30 (31)	32	96·9	14	16	46·7	—	—	—	2	6·7	—	—	—	—	1	—	—	—	—	1	28	93·3	11	4	6	5	2	—	—	—
		Control	*9*	*13*	*69·2*	*4*	*5*	*44·4*	—	—	—	*4*	*44·4*	*3*	—	—	—	—	*1*	—	—	—	—	*5*	*55·6*	*3*	—	—	*2*	—	—	—	—
		Total	39	45	86·7	18	21	46·2	—	—	—	6	15·4	3	—	—	—	1	1	—	—	—	1	33	84·6	14	4	6	7	2	—	—	—
	1927	Bonemeal	(34) 33	37	91·9	17	16	51·5	71·7	66·7	69·2	3	9·1	—	—	—	—	2	—	—	—	1	—	16	48·5	—	16	—	—	—	—	14	42·4
		Control	7 (8)	*13*	*61·5*	*4*	*3*	*57·1*	*71·0*	*77·0*	*73·6*	*1*	*14·3*	—	—	—	—	*1*	—	—	—	—	—	*1*	*14·3*	—	—	—	—	—	*1*	*5*	*71·4*
		Total	40	50	80·0	21	19	52·5	71·4	71·8	71·4	4	10·0	—	—	—	—	3	—	—	—	1	—	17	42·5	—	16	—	—	—	1	19	47·5
	1928	Bonemeal	27	31	87·1	16	11	59·3	74·5	73·9	74·3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	27	100
		Control	*5*	*9*	*55·6*	*3*	*2*	*60·0*	*69·0*	*71·0*	*69·8*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*5*	*100*
		Total	32	40	80·0	19	13	59·4	71·8	72·5	72·1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	32	100
	Total	Bonemeal	90	100	90·0	47	43	52·2	73·1	70·3	71·8	5	5·6	—	—	—	—	3	—	—	—	1	1	44	48·9	11	20	6	5	2	—	41	45·5
	Total	*Control*	*21*	*35*	*60·0*	*11*	*10*	*52·4*	*70·0*	*74·0*	*71·7*	*5*	*23·8*	*3*	—	—	—	*1*	*1*	—	—	—	—	*6*	*28·6*	*3*	—	—	*2*	—	*1*	*10*	*47·6*
		TOTAL	111	135	82·2	58	53	52·3	71·5	72·2	71·7	10	9·0	3	—	—	—	4	1	—	—	1	1	50	45·0	14	20	6	7	2	1	51	46·0
RED POLL.	1926	Bonemeal	28 (29)	37	78·4	13	15	46·4	—	—	—	1	3·6	—	—	—	—	—	—	—	—	—	1	27	96·4	9	5	5	5	3	—	—	—
		Control	*4*	*15*	*26·7*	*2*	*2*	*50·0*	—	—	—	*1*	*25·0*	—	—	—	—	—	—	—	—	—	*1*	*3*	*75·0*	—	—	*1*	*2*	—	—	—	—
		Total	32	52	61·5	15	17	46·9	—	—	—	2	6·3	—	—	—	—	—	—	—	—	—	2	30	93·7	9	5	6	7	3	—	—	—
	1927	Bonemeal	32 (33)	36	91·7	20	12	62·5	73·7	72·9	73·4	3	9·4	—	—	—	—	2	—	1	—	—	—	18	56·3	—	18	—	—	—	—	11	34·3
		Control	*8*	*12*	*66·7*	*6*	*2*	*75·0*	*76·7*	*60·5*	*72·6*	*1*	*12·5*	*1*	—	—	—	—	—	—	—	—	—	*2*	*25·0*	—	—	—	—	—	*2*	*5*	*62·5*
		Total	40	48	83·3	26	14	65·0	75·2	66·7	73·0	4	10·0	1	—	—	—	2	—	1	—	—	—	20	50·0	—	18	—	—	—	2	16	40·0
	1928	Bonemeal	27	30	90·0	11	16	40·7	68·6	61·9	64·9	1	3·7	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	26	96·3
		Control	*3*	*7*	*42·9*	*3*	*0*	*100*	*62·0*	—	*62·0*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*3*	*100*
		Total	30	37	81·1	14	16	46·7	65·3	61·9	63·5	1	3·3	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	29	96·7
	Total	Bonemeal	87	103	84·5	44	43	50·6	71·1	67·4	69·1	5	5·7	—	1	—	—	2	—	1	—	—	1	45	51·7	9	23	5	5	3	—	37	42·5
	Total	*Control*	*15*	*34*	*44·1*	*11*	*4*	*73·3*	*69·3*	*60·5*	*67·3*	*2*	*13·3*	*1*	—	—	—	—	—	—	—	—	*1*	*5*	*33·3*	—	—	*1*	*2*	—	*2*	*8*	*53·3*
		TOTAL	102	137	74·5	55	47	53·9	70·2	63·9	68·2	7	6·9	1	1	—	—	2	—	1	—	—	2	50	49·0	9	23	6	7	3	2	45	44·1
SUSSEX.	1926	Bonemeal	25	34	76·5	15	10	60·0	—	—	—	1	4·0	—	—	—	—	1	—	—	—	—	—	24	96·0	6	7	3	5	3	—	—	—
		Control	*9*	*16*	*56·3*	*7*	*2*	*77·8*	—	—	—	*3*	*33·3*	—	—	—	*2*	*1*	—	—	—	—	—	*6*	*66·7*	*2*	—	—	*2*	*2*	—	—	—
		Total	34	50	64·0	22	12	34·7	—	—	—	4	11·8	—	—	—	2	2	—	—	—	—	—	30	88·2	8	7	3	7	5	—	—	—
	1927	Bonemeal	30 (31)	34	91·2	17	13	56·7	71·9	69·9	69·0	—	—	—	—	—	—	—	—	—	—	—	—	17	56·7	—	17	—	—	—	—	13	43·3
		Control	*7*	*13*	*53·8*	*5*	*2*	*71·4*	*71·6*	*56·5*	*66·9*	*1*	*14·2*	*1*	—	—	—	—	—	—	—	—	—	*3*	*42·9*	—	—	—	—	—	*3*	*3*	*42·9*
		Total	37	47	78·7	22	15	59·4	71·8	63·2	68·0	1	2·7	1	—	—	—	—	—	—	—	—	—	20	54·1	—	17	—	—	—	3	16	43·2
	1928	Bonemeal	28	32	87·5	15	13	53·6	72·6	70·6	71·7	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	28	100
		Control	*6*	*9*	*66·7*	*3*	*3*	*50·0*	*71·7*	*60·3*	*66·0*	*1*	*16·7*	—	—	—	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	*5*	*83·3*
		Total	34	41	82·9	18	16	52·9	72·1	65·5	68·9	1	2·9	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	33	97·1
	Total	Bonemeal	83	100	83·0	47	36	56·6	72·3	70·2	70·4	1	1·2	—	—	—	—	1	—	—	—	—	—	41	49·4	6	24	3	5	3	—	41	49·4
	Total	*Control*	*22*	*38*	*57·9*	*15*	*7*	*68·2*	*71·6*	*58·4*	*66·4*	*5*	*22·7*	*1*	—	—	*2*	*1*	—	—	—	—	*1*	*9*	*40·9*	*2*	—	—	*2*	*2*	*3*	*8*	*36·4*
		TOTAL	105	138	76·1	62	43	59·0	71·9	64·3	68·4	6	5·7	1	—	—	2	2	—	—	—	—	1	50	47·6	8	24	3	7	5	3	49	46·7
Bonemeal Total for all Herds			344	400	86·0	174	170	50·6	70·2	68·0	68·8	13	3·8	—	1	—	—	7	—	1	1	1	2	169	49·1	32	85	18	20	11	3	162	47·1
Control Total for all Herds			*81*	*145*	*55·9*	*48*	*33*	*59·3*	*70·8*	*64·6*	*68·0*	*18*	*22·2*	*7*	—	*3*	*3*	*2*	*1*	—	—	—	*2*	*33*	*40·7*	*10*	—	*1*	*8*	*3*	*11*	*30*	*37·0*
GRAND TOTAL FOR ALL HERDS			425	545	78·0	222	203	52·2	70·4	66·3	68·4	31	7·3	7	1	3	3	9	1	1	1	1	4	202	47·5	42	85	19	28	14	14	192	45·2

In the column "Number of half-bred calves born," some figures are given in brackets (e.g., for Friesland 1926 and 1927, Red Poll 1926 and 1927, and Sussex 1927 calves). These figures correspond to the numbers given in Table No 1, which serves to show the fertility of the Basis Herd Cows. However, some of the calves were born dead. The figures not in brackets, given in the above column, denote the number of calves which were born alive in the experiment. All percentages are calculated on this figure.

Fertility, as far as the present experiment is concerned, must be considered from two aspects:—

1. The influence of bonemeal on fertility.
2. The influence of improved blood (Africander, Fries, Red Poll, Sussex), upon fertility, under a system of grading up native cows receiving a corrected mineral diet.

(1) *The Influence of Bonemeal upon Fertility.*

The available data show that fertility is markedly higher in bonemeal-fed cows than in the control cows receiving no bonemeal.

Over the period 18.3.25 to 30.6.28, the proportion of actual to possible number of calves (reckoning one calf to one cow per annum) works out at 87.3 per cent. for bonemeal-fed cows and 56.5 per cent. for the control cows. The figures are given in Table No. 3:—

TABLE NO. 3.

	Bonemeal Cows.	*Control Cows.*
Actual number of calves born in 1926, 1927, and 1928............	349	*82*
Possible number of calves in 1926, 1927, and 1928................	400	*145*
Percentage of actual to possible number of calves born in 1926, 1927, and 1928..	87·3	*56·5*

A closer study of the experimental data shows that, while the calving percentages of the bonemeal-fed cattle for the years 1926, 1927, and 1928 remained well over 80, those of the control cows fell from 59.7 per cent. to 51.6 per cent. The figures are given in table No. 4:—

TABLE NO. 4.

	Bonemeal Cows.	*Control Cows.*
Percentage of cows which calved in 1926........................	82·6	*59·7*
Percentage of cows which calved in 1927........................	92·9	*55·8*
Percentage of cows which calved in 1928........................	86·1	*51·6*

Table No. 5 further shows that, whereas the bonemeal-fed cows bred regularly, the control cows bred *very irregularly*.

Amongst the bonemeal-fed cows which remained in the experiment throughout the three years, 66.1 per cent. gave birth to three calves in the three years, whereas of the control cows which remained in the experiment for the same period, not a single one calved three times.

Some cows were in the experiment for only two years and others again for only one. In these cases the maximum number of calvings were, naturally, two and one respectively. A full record of the number of calvings of all cows in the experiment is given in table No. 1, but some of the more striking figures have been repeated in table No. 5:—

TABLE NO. 5.

	Bonemeal Cows.	*Control Cows.*
Number of cows which gave birth to three calves in three years....	72	*0*
Number of cows which could have had three calves in three years..	109	*20*
Percentage of cows which had three calves in three years..........	66·1	*0*
Number of cows which had two calves in two years..............	26	*5*
Number of cows which could have had two calves in two years....	30	*21*
Percentage of cows which had two calves in two years............	86·7	*23·8*
Number of cows which had one calf in one year..................	10	*25*
Number of cows which could have had one calf in one year........	13	*43*
Percentage of cows which had one calf in one year...............	76·9	*58·1*

The figures may also be given, perhaps in a more instructive form, in the following way:—

(*a*) Of the 109 *bonemeal*-fed cows which were in the experiment for *three seasons*:—

72 or 66.1 per cent. gave 3 calves in 3 years.
31 or 28.4 per cent. gave 2 calves in 3 years.
6 or 5.5 per cent. gave 1 calf in 3 years.

Of the 20 *control* cows which were in the experiment for *three seasons*:—

0 or 0 per cent. gave 3 calves in 3 years.
13 or 65 per cent. gave 2 calves in 3 years.
7 or 35 per cent. gave 1 calf in 3 years.

(*b*) Of the 30 *bonemeal*-fed cows which were in the experiment for *two seasons*:—

26 or 86.7 per cent. gave 2 calves in 2 years.
3 or 10 per cent. gave 1 calf in 2 years.
1 or 3.3 per cent. gave 0 calf in 2 years.

Of the 21 *control* cows which were in the experiment for *two seasons*:—

5 or 23.8 per cent. gave 2 calves in 2 years.
14 or 66.7 per cent. gave 1 calf in 2 years.
2 or 9.5 per cent. gave 0 calf in 2 years.

(*c*) Of the 13 *bonemeal*-fed cows which were in the experiment for *one season* only:—

10 or 76.9 per cent. gave 1 calf in 1 year.
3 or 23.1 per cent. gave 0 calf in 1 year.

Of the 43 *control* cows which were in the experiment for *one season*:—

25 or 58.1 per cent. gave 1 calf in 1 year.
18 or 41.9 per cent. gave 0 calf in 1 year.

If, finally, we glance at graph No. 1, which gives curves of the bi-monthly weights of bonemeal and control cows, over the period under review, it will be seen that while the curve for the bonemeal cows shows a definite upward tendency, the control weight curve shows a downward tendency.

Gradual loss in condition and body weight, and increasing irregularity in breeding, seem to be associated with the progressively lower fertility in the control cows.

Reviewing the breeding seasons 1926, 1927, and 1928, it would appear that during the 1926 season the control cows, which were then in fairly good condition, had a sufficient mineral reserve to produce quite a good calf crop. However, while these calves were being suckled, the drain on phosphates, through the milk, was so great that many of the cows were unable to breed the next year. These cows apparently did not possess the minimum phosphorus reserve necessary for breeding and hence skipped a year.

During the next season (1927), these cows may or may not have been able to build up their mineral reserve to the minimum mineral requirement for breeding. Those cows that were able to do so probably did breed in 1928, but the greater number apparently needed more than one year in which to recover. Hence these latter, together with practically all the control cows which gave calves two years in succession, failed to breed in 1928.

Since the available phosphorus in the pasture is far below the requirement of breeding stock, the result of successive calvings would seem to be a gradual reduction of the mineral reserves of the body which, in turn, will manifest itself either in the form of a deficiency disease, or in temporary, if not permanent, sterility.

(2) *The Influence of Improved Blood upon Fertility, under a System of Upgrading, on a Corrected Mineral Diet.*

Table No. 6 gives the available data:—

TABLE NO. 6.

	Africander.		Friesland.		Red Poll.		Sussex.	
	Bonemeal.	*Control.*	Bonemeal.	*Control.*	Bonemeal.	*Control.*	Bonemeal.	*Control.*
Percentage of cows which calved in 1926....................	82·9	*83·3*	96·9	*69·2*	78·4	*26·7*	76·5	*56·2*
Percentage of cows which calved in 1927....................	97·0	*42·9*	91·9	*61·5*	91·7	*66·7*	91·2	*53·8*
Percentage of cows which calved in 1928....................	79·0	*33·3*	87·1	*55·6*	90·0	*42·9*	87·5	*66·7*
Percentage of cows which calved in 1926, 1927, and 1928......	86·6	*60·5*	92·0	*62·8*	86·4	*41·1*	84·0	*57·9*

It is scarcely warranted to formulate any conclusions about the influence of improved blood upon fertility from the data quoted above. The results of many more years of breeding will have to be available, before anything like reliable deductions can be drawn. There is a further difficulty in interpreting the above data, and that is that no service register was kept during the years 1926, 1927, and 1928, and hence it is impossible to estimate in how far the fertility of the individual bulls which were used influenced the results obtained.

(*b*) Early Maturity.

In considering the influence of bonemeal feeding on early maturity, we must take into account:—

1. The influence of bonemeal feeding on the sexual maturity of the breeding stock.
2. The influence of bonemeal feeding on the body maturity.

It is evident that under each of these headings a comparative study of the four herds would be necessary.

(1) *The Influence of Bonemeal Feeding on the Sexual Maturity of Breeding Stock.*

For the purpose of collecting data in connection with the above question, the Basis Herd cows were unsuitable as nothing accurate was known of the ages of these animals, nor of their histories, prior to the beginning of the experiment.

The data so far available are limited to the service figures of the first batch of half-bred heifers which were put to the bulls from the 20th of February to the 20th of May, 1928.

Table No. 7.

	Africander.		Friesland.		Red Poll.		Sussex.	
	Bonemeal.	*Control.*	Bonemeal.	*Control.*	Bonemeal.	*Control.*	Bonemeal.	*Control.*
Total half-bred batch I heifers	6	*7 (4)*	11	*7 (6)*	9	*5*	6	*4*
Number of half-bred batch I heifers served................	5	*3*	11	*6*	9	*4*	6	*4*
Number of half-bred batch I heifers in calf on the 31/12/28	5	*3*	9	*3*	7	*4*	6	*4*

Before discussing the above table, a few points should be emphasized:—

(*a*) The above table gives the service results of heifers put to the bull at an age of 2 years and 3 months. It is quite probable, in fact it is quite certain, that many of the heifers would have conceived much earlier in life if they had been allowed to do so. Hence, the above data do not indicate the exact age at which the heifers became sexually mature. The table merely gives a comparative idea of the sexual maturity, expressed in number of services and conceptions, at the age of 2 to 2¼ years.

(*b*) The low figures in the Africander control column are incorrect in so far that 3 out of the 4 heifers which did not breed lost their dams within five months after birth. As a result of this they became stunted in their growth and should not be considered in the interpretation of these figures. The same applies to one of the Fries control heifers.

The corrected figures, for purposes of comparison, are given in brackets.

(*c*) The bulls used were active and never failed to cover a heifer when in season. The bull factor may thus be considered as equal for the four herds.

Discussion.—The figures quoted refer to so small a number of animals that it is unwise to draw any conclusions from them. The figures seem to indicate a slight beneficial effect of bonemeal feeding on the sexual maturity of animals at the age of 26 months. The respective figures for the bonemeal and control groups work out as follows:—

	Bonemeal.	*Control.*
Total No. of heifers	32	*19*
No. of heifers served	31	*17*
No. of heifers in calf	27	*14*
Per cent. heifers served	96.9 per cent.	*89.5 per cent.*
Per cent. heifers in calf ...	84.4 per cent.	*73.7 per cent.*

As to the effect of breed upon sexual maturity, the table tends to show that there is no appreciable difference between the four herds.

Very careful observations throughout the lives of the heifers, and especially during their breeding season, tend to show that the Africander grades reach sexual maturity later in life than do the Fries, Red Poll and Sussex grades. Of the imported breed grades, many bonemeal heifers were observed to be on heat at just over one year old, while of the indigenous breed grades, the heifers appeared to be only just fit for breeding at 26 months of age.

In time it is hoped to obtain sufficient data as to the influence of breed on sexual maturity, but since, in the present investigation, heifers are to be served at the age of 2 to 2¼ years, a special experiment will be necessary to determine the influence of bonemeal feeding upon the exact age of sexual maturity.

(2) *The Influence of Bonemeal Feeding on Body Maturity.*

This will be fully dealt with later on in this report when the influence of bonemeal feeding upon growth, increase in body weight, skeletal development, etc., is discussed.

(*c*) THE INFLUENCE OF BONEMEAL FEEDING UPON THE RETENTION OF CONSTITUTIONAL VIGOUR IN GRADES OF IMPROVED BREEDS.

As pointed out earlier in this article, experience has shown that where superior types are used to grade up native stock on mineral deficient pastures, the increased rate of growth and reproduction of the grades cannot be supported by the deficient pasture and, as a result, the constitutional vigour of the grades breaks down.

It is one of the main objects of the present investigation to ascertain whether the correction of the mineral deficiency in the pasture will result in the retention of unimpaired body vigour in grades throughout the process of grading up.

The very fact that within $3\frac{1}{4}$ years, 66.3 per cent. of the control basis herd cows and 22.2 per cent. of the first generation control calves have died, as compared with 8.9 per cent. and 3.8 per cent. respectively in the bonemeal cattle, is sufficient proof that bonemeal feeding has a remarkable effect upon constitutional vigour.

A casual comparison of bonemeal and control grades of equal ages is sufficient to impress the fact that the former are "true to type," whereas the latter are "off type."

The bonemeal grades are of good size for their age, are deep and wide of body, not too high on the legs, and of very good bone.

The control grades, on the other hand, are leggy, narrow and undeep of body, small for their age, and lack "substance" in their bony skeleton.

The control portion of the breeding experiment is doomed to an early end. Already 79 out of the 92 original control basis herd cows have either died or been discharged from the experiment; and of all the first generation control heifers, only 25 remain. This seems sufficient proof that on phosphorus deficient pastures, such as those of Bechuanaland, the grading up of native stock, without correcting the mineral diet, is bound to fail.

Whether the present constitutional excellence of the bonemeal grades will be retained in the second, third and fourth generations of grades remains to be seen. However, there is every reason to be optimistic in this respect.

(*d*) SEX OF FIRST GENERATION CALVES.

Before passing on to the result of the dietetic part of the experiments, it is of interest to compare the proportion of bull calves and heifer calves, not only in the bonemeal and control groups, but also in the different herds.

(1) *Percentage of Bull Calves and Heifers in the Bonemeal and Control Groups.*

From Table No. 8, it will be seen that of the total number of 344 half-bred bonemeal calves born, 174 were males and 170 were females. This shows a slight excess of males over females. In the control group there is a much more marked predominance of males, the figures being 48 bull calves and 33 heifers.

TABLE No. 8.

	Total Number of Half-bred Calves.	Number of Bull Calves.	Percentage of Bull Calves.	Number of Heifer Calves.
Bonemeal: Totals......	344	174	50·6	170
Control: Totals.........	*81*	*48*	*59·3*	*33*

(2) *Percentage of Bull Calves and Heifers in the Four Herds.*

Table No. 9 gives the numbers and percentages of half bred male and female calves, in each of the four grade herds.

The interesting point which is revealed by a study of this table is that, whereas in the Fries, Red Poll and Sussex grades (i.e. grades of imported breeds) there is a tendency to a preponderance of males over females, in the Africander grades (i.e. grades of an indigenous breed) the opposite appears to be the case.

Table No. 9.

	Africander.		Friesland.		Red Poll.		Sussex.	
	Bone-meal.	*Control.*	Bone-meal.	*Control.*	Bone-meal.	*Control.*	Bone-meal.	*Control.*
Number of bull calves born....	36	*11*	47	*11*	44	*11*	47	*15*
Number of heifer calves born...	48	*12*	43	*10*	43	*4*	36	*7*
Percentage of bull calves born..	46·5	*47·8*	52·2	*52·4*	50·6	*73·3*	56·6	*68·2*

This preponderance of heifers over bull calves might have been accounted for as the result of a particularly large number of heifers born during any one of the three seasons under review. This, however, is not borne out by the figures given in Table No. 10, in which details are given of the calving percentages of each breed for each of the three years.

Table No. 10.

	1926.		1927.		1928.	
	Bone-meal.	*Control.*	Bone-meal.	*Control.*	Bone-meal.	*Control.*
Number of Africander bull calves............	15	*7*	12	*4*	9	*0*
Percentage of Africander bull calves..........	51·7	*46·7*	37·5	*66·7*	39·1	*0*
Number of Africander heifers................	14	*8*	20	*2*	14	*2*

In conclusion, it would appear that there is a tendency for males to predominate in the grades of the Fries, Red Poll and Sussex breeds and for females to predominate in the Africander grades.

Amongst the grades of the imported breeds the inclination to male preponderance is most marked in the control groups, while the tendency to female dominance in the Africander (indigenous breed) grades is most marked in the bonemeal groups.

B. FEEDING RESULTS.

(*a*) The Influence of Bonemeal Feeding on the Birth and Weaning Weights of Calves.

Table No. 11 gives the birth and weaning weights of the first generation calves of each of the grade herds. Taking the bonemeal and control groups of calves, irrespective of herds, the average birth weight of a bonemeal calf works out at 68.8 lbs. and that of a control calf at 68.1 lbs.

From the above figures it seems justified to conclude that bonemeal feeding has no influence upon the *birth* weight of calves.

It might be thought that, as the phosphorus reserve of the body of a control cow gradually becomes smaller and smaller, less phosphorus would be available for the development of successive foetuses and that, as a consequence of this, the birth weight of the calves would decrease in proportion to the lessening phosphorus amounts available. However, it would seem that nature has made provision to prevent the birth of weaklings, as far as mineral constituents of the body are concerned. It would appear that unless a control cow has a phosphorus reserve sufficient for the full development of the foetus, she either does not come into season at all, or does not conceive. In any case, she does not breed.

Bonemeal, therefore, influences fertility but not the birth weight. Bonemeal-fed and control calves start life on an equal basis.

Nature, however, goes a step further. In order to ensure for each calf as good a start in life as possible, she has provided that the *quality of the milk* of the mother is not affected by phosphorus deficiency in the ration of the cow.

It has been shown chemically that qualitatively, *there is no difference in the mineral composition of the milk of bonemeal-fed and control cows* grazing on phosphorus deficient pastures.

If nature could also have ensured an equal *milk yield* for both bonemeal and control cows, the calves, up to weaning age at any rate, would have developed equally well.

This, however, is not the case and, as a result of the smaller milk yield (and consequently of the smaller total amount of phosphorus) of the control cows, there is a marked difference in average weights of the two groups at weaning time.

The average weight of the bonemeal calves was 409 lb. at weaning age (about 9 months) and for the control calves 351 lb.

If next we compare the average weights of the calves of the different herds, we find that the grades of the imported breeds weigh considerably more at birth than do the Africander (indigenous) grades. The figures are:—

	Bonemeal Calves.	*Control Calves.*
(*a*) Africander	64.0 lb.	66.7 lb.
(*b*) Fries	71.7 lb.	71.7 lb.
(*c*) Red Poll	69.2 lb.	67.3 lb.
(*d*) Sussex	70.4 lb.	66.5 lb.

TABLE NO. 11.

Born in.	Supplementary Ration.	Average Weight of Calves.									
		Africander.		Friesland.		Red Poll.		Sussex.		All Breeds.	
		At Birth.	At Weaning.	At Birth.	At Weaning.	At Birth.	At Weaning.	At Birth.	At Weaning.	At Birth.	At Weaning.
1926....	Bonemeal..........	—	433	—	433	—	375	—	422	—	415·8
	Control............	—	*378*	—	*340*	—	*357*	—	*342*	—	*354·3*
1927....	Bonemeal..........	65·6	393	69·2	417	73·4	381	69·0	424	69·3	403·8
	Control............	*69·5*	*348*	*73·6*	*359*	*72·6*	*356*	*66·9*	*424*	*70·7*	*371·8*
1928....	Bonemeal..........	62·5	375	74·3	436	64·9	387	71·7	433	68·4	407·8
	Control............	*64·0*	*280*	*69·8*	*336*	*62·0*	*319*	*66·0*	*378*	*65·5*	*328·3*
1926–28.	Bonemeal..........	64·0	400	71·7	429	69·2	381	70·4	426	68·8	409·0
	Control............	*66·7*	*335*	*71·7*	*345*	*67·3*	*344*	*66·5*	*381*	*68·1*	*351·3*

The fact that the Africander grade calves weigh less at birth than do the grade calves of imported breeds may possibly be explained as follows:—

Ever since the first colonists found the progenitors of the present breed in the possession of the Hottentots, and probably long before that, the Africander cattle have always been "veld animals" and have had to live on phosphorus deficient pastures in many parts of the country.

Adaptation to this limiting factor in the nutrition has produced such characteristics as small light bone, light body weight, late maturity, etc.

These points are characteristic of the breed and possess quite as potent an influence on the Africander grades as the bigger bone, heavier body weight, earlier maturity, etc., of the imported breeds have on their grades.

Although in our experiments this limiting factor has been corrected, systematic selection and breeding, under the correct nutritional conditions, will probably have to be carried out for many generations before the Africander will reach the level of breeds which were developed in areas of phosphorus-rich pastures.

In the first few generations we may expect quite an appreciable difference in body weight, growth, skeletal development, early maturity, etc., between the Africander and imported breed grades.

However, it is probable that, in time, these differences will become less and less marked, until finally the Africander can compete in all respects with the imported breeds.

Table No. 11 further shows that although the difference in weight between bonemeal and control calves of each herd at birth is at most 4 lb., the difference at weaning time is at least 37 lb. in favour of the bonemeal calves. The biggest difference is noticed in the Fries (84 lb.), while the second biggest difference is found in the Africander, in which breed at birth the control calves weighed on an average 2.7 lb. more than the bonemeal calves.

Finally, it is of interest to note that, according to the available data, the development of the Africander bonemeal calves appears to be quite as fast as that of the Red Poll calves during the first nine months of life.

(*b*) The Influence of Bonemeal Feeding on Growth.

For the study of the influence of bonemeal feeding on growth, the figures relating to batch 1 (1926) half-bred grades are the most complete and hence the most useful.

Under the previous heading the factors which affect the general development of bonemeal and control calves up to the time of weaning were discussed. In the present section the results of bonemeal feeding from weaning onwards, i.e. from the time that the weaners become totally dependent upon the natural pasture for their sustenance, will be discussed.

Table No. 12 gives bi-monthly average weights of all the half-bred bonemeal and control calves, irrespective of breed, from six months to two years and six months of age.

TABLE No. 12.

	6 months old.	8 months old.	10 months old.	12 months old.	14 months old.	16 months old.	18 months old.	20 months old.	22 months old.	24 months old.	26 months old.	28 months old.	30 months old.	32 months old.	34 months old.
	6/7/26.	3/9/26.	3/11/26.	4/1/27.	4/3/27.	5/5/27.	6/7/27.	5/9/27.	6/11/27.	6/1/28.	5/3/28.	5/5/28.	5/7/28.	5/9/28.	5/11/28.
Half-bred batch I bone-meal calves..........	378	420	478	564	659	701	746	728	722	804	932	982	976	913	915
Half-bred batch I control calves..............	*330*	*368*	*401*	*473*	*552*	*591*	*612*	*585*	*577*	*637*	*730*	*741*	*733*	*677*	*679*
DIFFERENCE...........	48	52	77	91	107	110	134	143	145	167	202	241	243	236	236

As shown in the preceding chapter, the average birth weights of bonemeal and control calves are practically identical. After six months, however, there is already a difference of 48 lb. in favour of the bonemeal calves. This advantage in body weight is maintained and increased throughout the period under consideration, until at 30 months old the bonemeal animals weigh 243 lb. or 33.2 per cent. more than the controls.

Bonemeal feeding starts directly after the calves are weaned at nine months of age. In this case, therefore, bonemeal was fed over a period of 21 months. The increase in the difference in weight between bonemeal-fed calves and control calves, which was 48 lb. at six months and 243 lb. at 30 months, was obtained at an approximate cost of 8s. 9d. per head, or just about ½d. per pound live weight. It is quite evident that this extra outlay was a very profitable one, especially as the quality of bonemeal beef is much better than that of control beef and, hence, commands a better price.

Graph No. 2 gives comparative weight curves for the bonemeal and control groups of half-bred calves, from six months up to 30 months of age. As the calves were only weaned at nine months of age, the part of the curves, July to November, 1926, represents weights of calves which were still receiving their mothers' milk. From November onwards, however, the calves were totally dependent upon the natural pasture for their sustenance.

Table No. 13 gives for both bonemeal and control calves the average percentage increase or decrease in body weight between two successive weighings. An increase in body weight is indicated with a plus sign and a decrease in weight with a minus sign, in front of the percentage values.

In general, the curves of graph No. 2 illustrate the same changes as the figures given in table No. 12. However, a closer study of the curves, especially in conjunction with a study of the figures given in table No. 13, reveals some very interesting points.

1. It will be noticed that, whereas throughout the winter of 1926 both the bonemeal and control groups developed rapidly in weight, in 1927 both groups actually decreased in weight during the same period of the year.

This is probably to be explained by the fact that in 1926 the calves still received a certain amount of milk from their dams, and that the growth impetus is greater in 6 months old tollies and heifers than in the same animals at 18 months of age. This is clearly shown in table No. 13 when the percentage increases in weight during the two periods July, 1926, to July, 1927, and July, 1927, to July, 1928, are compared.

2. The first rains fell in October, 1926 (1.10 inches). This was sufficient to allow the veld grasses to revive, and it is noticeable how both groups of calves reacted immediately to the improved pasture conditions. Whereas the percentage increase in body weight for bonemeal and control groups over the period September-November, 1926, was 13.8 and 9.0 respectively, these percentages jumped to 18 in both groups over the period November, 1926, to January, 1927.

3. It will be noticed from table No. 13 that the highest percentages of increase in body weights during the seasons 1926/1927

TABLE NO. 13.

	1926.		1927.						1928.					
	July–Sept.	Sept.–Nov.	Nov.–Jan.	Jan.–March.	March–May.	May–July.	July–Sept.	Sept.–Nov.	Nov.–Jan.	Jan.–March.	March–May.	May–July.	July–Sept.	Sept.–Nov.
	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Half-bred batch I bonemeal.....	+11·1	+13·8	+18·0	+16·8	+6·4	+6·4	—2·4	—0·8	+11·4	+15·9	+5·4	—0·6	—6·5	+0·2
Half-bred batch I control.........	*+11·5*	*+9·0*	*+18·0*	*+16·7*	*+7·1*	*+3·6*	*—4·4*	*—1·4*	*+10·4*	*+14·6*	*+1·5*	*—1·1*	*—7·6*	*+0·3*

and 1927/1928 occur early in summer during the time of *young* green grass. This holds true for the control as well as for the bonemeal animals.

Theiler, Green, and Du Toit (1924) have shown that the highest percentage of available P_2O_5 in pasture is found in the *young* green leaf stage. Hence it appears as if maximum increase in weight in cattle is directly associated with a high percentage of available P_2O_5 in the veld grasses.

4. Graph No. 2 and table No. 13 show that in both the bonemeal and control groups increase in weight in 1927 continued up to July or mid-winter, while in 1928 the increase in weight stopped in May or two months sooner.

The reason for this is to be found in the difference in quality of the late summer pasture in the two years under consideration. This is a point of considerable importance and warrants a fuller discussion.

Graph No. 3 gives the rainfall for the seasons 1926/1927 and 1927/1928.

It will be seen that the rainy season 1926/1927 began well with nearly three inches in December, which is above the normal for that month for Bechuanaland. In January only 1.9 inches fell, which is rather low, and during February, which usually is the month of greatest precipitation, only half an inch was registered.

March, 1927, was very wet indeed.

As a result of the dry (and particularly hot) months of January and February, 1927, the growth of the veld was badly checked and temporarily stopped. The growth after the good December rains had been upward growth or haulm growth. When the period of drought was broken in the middle of March, the pasture again started to grow, but now the grass grew, not in an upward direction with haulm formation, but in sideward growth with stooling and the formation of a large number of soft and short leaves near to the ground.

These leaves never became very fibrous and in autumn dried up and became curly. This " stool " or " pod " grass is the best winter pasture for cattle and, when abundant, ensures a long growing season and a good winter. It was probably on account of this condition of the pasture that the weights continued to increase up to July, 1927. The 1927/1928 rainy season may be described as having been a continuous one. The rain which fell in October, November and December, although insufficient to make the grass shoot into haulms, sufficed for the veld to revive from its winter condition and supply fair grazing. This resulted in an appreciable increase in weight in all the cattle.

With the plentiful January, February and March rains the pasture shot into haulm and seed and, because of its uninterrupted growth, very little low " stool " or " pod " grass was formed.

As a consequence of this, the cattle did not do so well, although there was actually much more grass than during the previous season. The animals started to drop in condition sooner, and did not winter nearly as well as in the preceding year.

It would appear, therefore, that continuous rainy seasons result in very active upward growth and consequently in poor " winter veld," whilst a discontinuous rainy season results in good " stooling " and in the production of excellent " winter veld," provided the total precipitation is not abnormally low or the periods of drought abnormally long.

5. The effect of good and bad winter veld is also reflected in a greater loss of weight during winter in years of bad winter veld than in years of good veld.

In 1927 the decrease in weight continued over four months (July to November); in 1928 it continued over six months (May to November). Table No. 14 gives the percentages of loss in body weight between successive weighings for the 1927 and 1928 winter seasons.

TABLE No. 14.

	1927.			1928.			
	July–Sept.	Sept.–Nov.	Total percentage loss over 4 months.	May–July.	July–Sept.	Sept.–Nov.	Total percentage loss over 6 months.
	%	%	%	%	%	%	%
Half-bred batch I bonemeal calves........	—2·4	—0·8	—3·2	—0·6	—6·5	+0·2	—6·8
Half-bred batch I control calves..........	*—4·4*	*—1·4*	*—5·7*	*—1·1*	*—7·6*	*+0·3*	*—8·4*

It is seen that in the bonemeal and control lots the percentage loss in weight in 1928 was 6.8 and 8.4 respectively, while in 1927 the figures were 3.2 and 5.7.

6. Graph No. 2 further brings out the fact that, during the winter months, the bonemeal cattle fall less in body weight than do the control cattle. Thus, over the period July to November, 1927, the bonemeal cattle lost 24 lb. or 3.2 per cent. of their body weight per head, while the control cattle lost on an average 35 lb. or 5.7 per cent.

Table No. 14 shows that the same happened in 1928, when the average loss in the bonemeal animals was 6.8 per cent. and the average loss among the controls 8.4 per cent.

So much for the influence of bonemeal feeding on growth, in general.

We must next consider:—

The effect of improved blood upon growth, under a system of bonemeal feeding.

Again the data of the half-bred batch I cattle are the most complete, and will be used for this comparative study.

Table No. 15.

Age of Animals in Months............	6	8	10	12	14	16	18	20	22	24	26	28	30
Date of Weighing....................	6/7/26.	3/9/26.	3/11/26.	4/1/27.	4/3/27.	5/5/27.	6/7/27.	5/9/27.	6/11/27.	6/1/28.	5/3/28.	5/5/28.	5/7/28.
Africander half-bred batch I bonemeal calves............................	389	419	471	569	655	704	738	721	715	794	906	941	952
Africander half-bred batch I control calves	*358*	*372*	*388*	*462*	*535*	*571*	*585*	*558*	*553*	*614*	*715*	*702*	*712*
Friesland half-bred batch I bonemeal calves............................	389	440	493	567	661	683	739	730	724	808	928	961	950
Friesland half-bred batch I control calves..	*332*	*388*	*420*	*472*	*541*	*576*	*602*	*564*	*564*	*633*	*721*	*750*	*727*
Red Poll half-bred batch I bonemeal calves............................	326	380	445	540	639	685	732	712	701	770	917	980	972
Red Poll half-bred batch I control calves..	*319*	*367*	*418*	*501*	*586*	*635*	*652*	*637*	*620*	*677*	*770*	*786*	*769*
Sussex half-bred batch I bonemeal calves	408	442	501	578	679	731	774	747	747	844	975	1,047	1,028
Sussex half-bred batch I control calves.....	*312*	*344*	*377*	*455*	*544*	*580*	*608*	*569*	*569*	*622*	*713*	*727*	*724*

Table No. 15 gives the bi-monthly average weight of the bonemeal and control groups of each of the four herds from the age of 6 months up to 2½ years old.

Let us first consider the *bonemeal groups* of the four herds. Graph No. 4 gives the weight curves for these groups of animals from 6 to 30 months old.

If we study graph No. 4 in conjunction with table No. 15, we see that the Sussex grades are the heaviest at 6 months old (408 lb.) and remain so up to the end of the curve. In fact, they gradually draw away from the other herds and end up at 2½ years of age with an average weight of 1,028 lb. In two years' time, their average weight has increased from 408 lb. to 1,028 lb., or 152 per cent. (Compare Figures 60, 61, 63, 64, 65, and 66.)

In the Sussex, as a true beef breed, this quick growth and rapid increase in weight could have been expected.

From the point of view of early maturity the Red Poll grades might have been expected to come next. Graph No. 4 shows that at 6 months old the Red Poll grades weighed on an average only 326 lb., or 63 lb. less than the grades of any of the other herds. It is difficult to explain this low average weight, especially as the average Red Poll birth weight is quite as high as the average birth weights of the grades of the other breeds.

Studying the Red Poll curve, we see that it overtakes the Africander and Fries curves fairly rapidly and, at 2½ years, actually lies well above them with an average of 22 lb. more than either of these two breeds. This rate of growth is remarkably fast. In 2 years the average weight increases from 326 lb. to 972 lb., i.e. a 198 per cent. increase. (See Figures 51, 52, 54, 55, 56, and 57.)

The very low weights at 6 months old and the remarkable rate of growth of the Red Poll grades will be discussed in greater detail further on in this article.

The Fries, or true dairy type grades, show a 144 per cent. increase in weight in two years, which is the rate which could have been expected in dairy stock, compared with that of dual-purpose and beef types. (See figures 40, 41, 42, 43, 46, 47 and 48.)

It may be pointed out that, on the 6th July, 1926, and on the 5th July, 1928, the Africander and Fries half-bred grades weighed exactly the same. The increase in weight for the Africander is the same as that of the Fries, i.e. 144 per cent., in two years, but if we follow the two curves carefully we see that throughout their course the Fries has a slight advantage.

Of the four herds the Africander herd is the slowest in growth. (See figures 31, 32, 35,36 and 37.)

A comparison of the rate of growth, as indicated by weight records, shows that the bonemeal grades of imported breeds are faster in growth and development than bonemeal grades of the indigenous breed. The reason for this has been discussed earlier in this paper.

Graph No. 5 gives the comparative weight curves for the *control groups* of the four herds.

It will be noticed that the Africander is again the poorest of the four herds, the percentage increase being only 99 per cent., notwithstanding the fact that, at the age of 6 months, they weighed on an average 26 lb. more than the grades of any of the other herds. (See figures 33, 34, 38 and 39.)

The Fries shows a percentage increase of 119 per cent. (Figures 44, 45, 49, and 50), the Red Polls of 141 per cent. (Figures 54, 59, and 60) and the Sussex of 132 per cent. (Figures 62, 67, and 68).

Here again the grades of imported breeds do better than the Africander grades and, as in the bonemeal groups, the Red Poll grades show the greatest percentage increase in weight.

The figures so far discussed may, for easy reference, be tabulated as follows:—

TABLE 16.

	Africander.		Friesland.		Red Poll.		Sussex.	
	Bonemeal.	*Control.*	Bonemeal.	*Control.*	Bonemeal.	*Control.*	Bonemeal.	*Control.*
	lb.	lb.	lb.	lb.	lb.	lb.	lb.	lb.
Weight batch I at 6 months old	389	*358*	389	*332*	326	*319*	408	*312*
Weight batch I at 30 months old	952	*712*	950	*727*	972	*769*	1,028	*724*
Difference in Weight........,.	563	*354*	561	*395*	646	*450*	620	*412*
	%	%	%	%	%	%	%	%
Percentage increase in weight	144	*99*	144	*119*	198	*141*	152	*132*
	lb.		lb.		lb.		lb.	
Difference in weight between bonemeal and control at 6 months....................	31		57		7		96	
Difference in weight between bonemeal and control at 30 months...................	240		223		203		304	
	%		%		%		%	
Percentage difference in weight between bonemeal and control at 30 months...............	33·7		30·7		26·4		42·0	

As stated above, both bonemeal and control groups of the Red Poll grades show a remarkable rate of growth as compared with the grades of the other herds. From the figures in table 16 it is seen that, at six months, there was only 7 lb. difference between the average weights of bonemeal and control Red Polls and that at 30 months this difference had increased to 203 lb.

For the other herds the differences in weight between the bonemeal and control groups at $2\frac{1}{2}$ years of age are respectively:—

(*a*) Africander 240 lb. or 33.7 per cent.
(*b*) Fries 223 lb. or 30.7 per cent.
(*c*) Sussex 304 lb. or 42.0 per cent.

Graphs 6, 7, 8, and 9 give the weight curves of the bonemeal and control groups, respectively, of each of the four herds.

A study of these graphs reveals interesting differences between the four groups. The Red Poll graph immediately arrests the attention because of the fact that the bonemeal and control curves run much closer together than in the other graphs.

The superior growing powers of the Red Poll grades seem to have minimized the effect of bonemeal feeding.

This effect is most marked in the Sussex grades, while in the Africander and Fries grades the effect seems to be more or less the same.

(*c*) The Influence of Bonemeal Feeding on the Quantity and Quality of Beef.

In the previous section tables and graphs were given to show that bonemeal feeding has a remarkable influence on growth and increase in weight. The figures quoted referred to batch 1 of the half-bred grades, irrespective of sex.

As a rule the majority of slaughter stock in South Africa are oxen, and hence, to study the effect of bonemeal on the quality and quantity of beef, five bonemeal and two control tollies were selected from each of the four herds (batch 1) for a special slaughter test. The small number of controls was unavoidable on account of the reduction in numbers and the unfertility of the control herds.

Except for the feeding of bonemeal, all the tollies were treated alike in every way; they did not receive any extra feeding, and were dependent on the natural pasture exclusively for their sustenance.

When selected, the tollies were two years and two months old, and it was decided not to slaughter them at any definite age, but to wait until the majority of the bonemeal animals had reached an average live weight of approximately 1,200 lb.

Table 17 gives the average bi-monthly weights of the bonemeal and control groups of each herd from the 1st November, 1926, to the 1st November, 1928, i.e. from the age of six months to 30 months:

It will be seen from the above table that none of the bonemeal oxen had reached the prescribed body weight for slaughtering purposes by November, 1928, and hence we have to confine the present discussion to the increase in weight and growth. The effect of bonemeal upon such factors as "dressing percentage," quality of the beef, etc., will have to be considered at a later date.

Sufficient evidence has already been given to prove the beneficial effect of bonemeal feeding on growth in general; hence the discussion in this section may be limited to a comparative study of the effect of improved blood upon growth and increase in weight, under a system of bonemeal feeding.

Let us again compare, first of all, the bonemeal tollies of the four herds; graph No. 10 gives the comparative weight curves.

Table No. 18 gives the percentage increase in weight between the bi-monthly weighings of the bonemeal and control groups of the four herds:

TABLE 17.

	1926.			1927.						1928.					
	July.	Sept.	Nov.	Jan.	March.	May.	July.	Sept.	Nov.	Jan.	March.	May.	July.	Sept.	Nov.
Africander half-bred batch I bonemeal calves	400	433	479	581	678	726	760	739	736	782	942	1,015	1,010	935	909
Africander half-bred batch I control calves	*444*	*460*	*505*	*587*	*647*	*677*	*713*	*683*	*681*	*727*	*810*	*824*	*843*	*770*	*730*
Friesland half-bred batch I bonemeal calves	414	490	545	604	687	726	798	768	762	814	968	1,028	1,023	950	925
Friesland half-bred batch I control calves	*336*	*372*	*380*	*433*	*505*	*510*	*562*	*522*	*529*	*577*	*675*	*713*	*700*	*647*	*594*
Red Poll half-bred batch I bonemeal calves	344	404	514	625	724	785	821	793	792	820	1,023	1,099	1,090	992	964
Red Poll half-bred batch I control calves	*301*	*362*	*398*	*487*	*575*	*602*	*637*	*604*	*595*	*623*	*750*	*825*	*732*	*671*	*659*
Sussex half-bred batch I bonemeal calves	401	460	514	610	712	753	805	761	771	816	1,000	1,098	1,045	976	955
Sussex half-bred batch I control calves	*258*	*355*	*417*	*496*	*571*	*615*	*634*	*592*	*567*	*622*	*736*	*750*	*738*	*666*	*655*

TABLE 18.

	1926.		1927.						1928.						Percentage total increase in weight, July, 1926–July, 1928.
	July–Sept.	Sept.–Nov.	Nov.–Jan.	Jan.–March.	Mar.–May.	May–July.	July–Sept.	Sept.–Nov.	Nov.–Jan.	Jan.–March.	Mar.–May.	May–July.	July–Sept.	Sept.–Nov.	
Africander half-bred batch I bonemeal tollies......	+8·3	+10·6	+21·3	+16·7	+7·1	+4·7	—2·8	—0·4	+6·3	+20·5	+7·7	—0·5	—7·4	—2·8	127·3
Africander half-bred batch I control tollies.........	*+3·6*	*+9·8*	*+16·2*	*+10·2*	*+4·6*	*+5·3*	*—4·2*	*—0·3*	*+6·8*	*+11·4*	*+1·7*	*+2·3*	*—8·7*	*—5·2*	*64·4*
Friesland half-bred batch I bonemeal tollies......	+18·4	+11·2	+10·8	+13·7	+5·7	+9·9	—3·8	—0·8	+6·8	+18·9	+6·2	—0·5	—7·1	—2·6	123·4
Friesland half-bred batch I control tollies..........	*+10·7*	*+2·2*	*+13·9*	*+16·6*	*+1·0*	*+10·2*	*—7·1*	*+1·3*	*+9·1*	*+17·0*	*+5·6*	*—1·8*	*—7·6*	*—8·2*	*76·8*
Red Poll half-bred batch I bonemeal tollies......	+17·4	+27·2	+21·6	+15·8	+8·4	+4·6	—3·4	—0·1	+3·6	+24·7	+7·4	—0·8	—9·0	—2·8	180·2
Red Poll half-bred batch I control tollies..........	*+20·2*	*+9·9*	*+22·4*	*+18·1*	*+4·7*	*+5·8*	*—5·2*	*—1·5*	*+4·7*	*+20·4*	*+10·0*	*—11·3*	*—8·3*	*—1·8*	*118·9*
Sussex half-bred batch I bonemeal tollies.......	+14·7	+11·7	+18·7	+16·7	+5·8	+6·9	—5·5	+1·3	+5·8	+22·5	+9·8	—4·8	—6·6	—2·2	138·2
Sussex half-bred batch I control tollies..........	*+37·6*	*+17·5*	*+18·9*	*+15·1*	*+7·7*	*+3·1*	*—6·6*	*—4·2*	*+9·7*	*+18·3*	*+1·9*	*—1·6*	*—9·8*	*—1·7*	*153·9*

Making a combined study of tables 17 and 18 and graph No. 10, the following points are worthy of note:

The first point to be emphasized is the wonderful power of growth of the Red Poll grades (see Figures 51, 54, and 56). At the age of five months they weighed on an average 344 lb. or 56 lb. less than any other breed. At two years and six months the average weight is 964 lb., or 9 lb. more than the tollies of the next heaviest herd (i.e. Sussex 955 lb.). The increase in weight in the two years is, therefore, 180.2 per cent. or 42.0 per cent. more than the next best batch (i.e. Sussex) (Figures 60, 63 and 65).

It will be remembered that, in discussing table No. 15 and graph No. 4, the low average weight of the batch I Red Poll calves at six months of age was remarked upon, and that no explanation could be given why the Red Polls showed such a very high increase in weight (198 per cent.) as compared with the other herds (Sussex 152 per cent. and Fries and Africander 144 per cent.).

With the further figures of tables No. 17 and 18 now available, it seems justified to attribute the apparently very slow development during the first six to nine months of life and the remarkable impetus to growth thereafter definitely to the influence of Red Poll blood.

With our present knowledge, it is impossible to state why the six months old Red Poll grades should be so much lighter in body weight than the grades of the other herds at the same age; the fact, however, that from weaning onwards these animals develop more rapidly than any of the other herds, is of sufficient importance to be emphasized here. It is also of interest to ascertain whether this superior growth impetus manifests itself throughout the period under review or, if it is not continuous, at what period or age it is most pronounced.

Table No. 19 gives for the bonemeal control groups of each herd the average percentage increase or decrease in weight for the four periods:—

1. July, 1926, to July, 1927.
2. July, 1927, to November, 1927.
3. November, 1927, to May, 1928.
4. May, 1928, to November, 1928.

Period No. 1 extends over 12 months (i.e. tollies from six to 18 months old). During this period, there was a continuous increase in weight. The second period covers the winter of 1927, and lasted four months. It shows a continuous decrease in weight. The third period covers the summer of 1927/28 with a steady increase in body weight; and the last period again represents the winter of 1928, and shows a gradual loss in weight.

It was noticed that in 1927 the animals increased in weight up to July, while in 1928 they did so only up to May. As explained before, this was due to a very good summer in 1927 and a very bad one in 1928.

It will be seen from the above table that, during the period July, 1926, to July, 1927, the average percentage increase in weight of the Red Poll bonemeal grades was much higher than that of the bonemeal grades of the other three herds. During the period July, 1927, to November, 1927, the loss of weight was well below the average loss for the other three herds, and during the third period (November, 1927, to May, 1928) the percentage increase in weight was second only to that of the Sussex grades.

Hence the growth impetus of the Red Poll grades appears to be quite as strong as that of the other herds throughout the period from the age of six to the age of 30 months.

It is, however, much greater than that of the grades of the other herds between the ages of six and 18 months. Within this period again the power to grow appears to be most pronounced during the spring months.

TABLE 19.

	Average bi-monthly increase in weight, July, 1926–July, 1927.	Average bi-monthly decrease in weight, July, 1927–Nov., 1927.	Average bi-monthly increase in weight, Nov., 1927–May, 1928.	Average bi-monthly decrease in weight, May, 1928–Nov., 1928.
	%	%	%	%
Africander half-bred batch ! bonemeal tollies..................	+11·5	—1·6	+11·5	—3·6
Africander half-bred batch I control tollies....................	*+8·3*	*—2·3*	*+6·6*	*—3·9*
Friesland half-bred batch I bonemeal tollies..................	+11·6	—2·3	+10·6	—3·4
Friesland half-bred batch I control tollies........................	*+9·1*	*—2·9*	*+10·6*	*—5·9*
Red Poll half-bred batch I bonemeal tollies..................	+15·8	—1·8	—11·9	—4·2
Red Poll half-bred batch I control tollies........................	*+13·5*	*—3·3*	*+11·7*	*—7·1*
Sussex half-bred batch I bonemeal tollies..................	+12·4	—2·1	+12·7	—4·5
Sussex half-bred batch I control tollies........................	*+16·6*	*—5·4*	*+10·0*	*—4·4*

From graph No. 10, it would appear as if the Africander tollies (Figures 31, 35 and 37) are inferior to the tollies of imported breeds in respect of rate of growth and development. Table No. 18 shows that this is not so. The Fries tollies (Figures 40, 42, 46, and 48) show a total increase in weight of 123.4 per cent., while the Africander tollies show a 127.3 per cent. increase.

A comparison between Fries and Africander tollies in this respect is, however, not quite fair. In South Africa the Fries breed is looked upon as a definite dairy breed, while the Africander is being developed more and more along beef lines; therefore Africander tollies are expected to mature more quickly than tollies of the dairy type.

TABLE

Animal.	Animal number.	Herd.	Date of birth.	Ration.	Date when measured.	Weight.	Body length.
Basis herd cow........	1359	Africander...	—	Bonemeal.....	17/3/27	840	141·0
Half-bred batch I heifer	1841	,, ...		,, ...	17/3/27	700	132·8
,, ,, ,,	1841	,, ...		,, ...	10/3/28	950	142·7
						250	9·9
Basis herd cow.........	*1308*	*Africander...*	—	*Control......*	*18/3/27*	*711*	*139·8*
Half-bred batch I heifer	*2683*	,, ...		,, ...	*18/3/27*	*440*	*113·3*
,, ,, ,,	*2683*	,, ...		,, ...	*10/3/28*	*625*	*126·8*
						185	*13·5*
Basis herd cow........	1301	Africander...	—	Bonemeal....	18/3/27	1040	153·3
Half-bred batch I heifer	1850	,, ...		,, ...	18/3/27	706	131·5
,, ,, ,,	1850	,, ...		,, ...	10/3/28	965	140·7
						259	9·2
Basis herd cow.........	*1620*	*Africander...*	—	*Control......*	*17/3/27*	*684*	*132·3*
Half-bred batch I heifer	*1847*	,, ...		,, ...	*17/3/27*	*545*	*121·8*
,, ,, ,,	*1847*	,, ...		,, ...	*10/3/28*	*670*	*125·2*
						125	*3·4*
Basis herd cow........	1307	Friesland....	—	Bonemeal....	17/3/27	960	145·7
Half-bred batch I heifer	2013	,, ...		,, ...	17/3/27	593	127·5
,, ,, ,,	2013	,, ...		,, ...	10/3/28	891	146·3
						298	18·6
Basis herd cow.........	*1295*	*Friesland....*	—	*Control......*	*17/3/27*	*595*	*133·8*
Half-bred batch I heifer	*1920*	,, ...		,, ...	*17/3/27*	*464*	*116·2*
,, ,, ,,	*1920*	,, ...		,, ...	*10/3/28*	*582*	*127·3*
						118	*11·1*
Basis herd cow........	1373	Friesland....	—	Bonemeal....	17/3/27	1268	163·8
Half-bred batch I heifer	1904	,, ...		,, ...	17/3/27	691	130·5
,, ,, ,,	1904	,, ...		,, ...	10/3/28	920	147·5
						229	17·0
Basis herd cow.........	*1354*	*Friesland....*	—	*Control......*	*17/3/27*	*712*	*142·7*
Half-bred batch I tollie	*1978*	,, ...		,, ...	*17/3/27*	*642*	*127·2*
,, ,, ,,	*1978*	,, ...		,, ...	*10/3/28*	*815*	*142·0*
						173	*14·8*
Basis herd cow........	1411	Red Poll....	15/1/26	Bonemeal....	17/3/27	926	144·8
Half-bred batch I heifer	2136	,, ...		,, ...	17/3/27	689	128·2
,, ,, ,,	2136	,, ...		,, ...	10/3/28	961	144·2
						272	16·0
Basis herd cow.........	*1234*	*Red Poll....*	*1/2/26*	*Control......*	*17/3/27*	*750*	*150·2*
Half-bred batch I tollie	*2024*	,, ...		,, ...	*17/3/27*	*510*	*118·2*
,, ,, ,,	*2024*	,, ...		,, ...	*10/3/28*	*680*	*138·2*
						170	*20·0*
Basis herd cow........	1356	Sussex.......	24/12/25	Bonemeal....	17/3/27	965	139·3
Half-bred batch I heifer	1905	,, ...		,, ...	17/3/27	600	125·7
,, ,, ,,	1905	,, ...		,, ...	10/3/28	1000	141·5
						400	15·8
Basis herd cow.........	*1402*	*Sussex.......*	*14/1/26*	*Control......*	*17/3/27*	*755*	*140·8*
Half-bred batch I tollie	*1975*	,, ...		,, ...	*17/3/27*	*624*	*124·8*
,, ,, ,,	*1975*	,, ...		,, ...	*10/3/28*	*828*	*138·0*
						204	*13·2*
Basis herd cow........	1480	Sussex.......	29/12/25	Bonemeal....	17/3/27	924	146·2
Half-bred batch I heifer	1917	,, ...		,, ...	17/3/27	673	119·8
,, ,, ,,	1917	,, ...		,, ...	10/3/28	990	124·2
						317	4·4

No. 20.

Body height.	Height at hook-bones.	Depth of chest.	Width of chest.	Width between hook-bones.	Width between thirls.	Length of croup.	Width between pin-bones.	Heart girth.	Length of head.	Width between eyes.
117·0 117·3 130·5	121·7 117·8 134·1	65·3 60·2 67·2	36·0 32·0 37·5	48·2 41·0 47·7	41·7 36·8 39·3	45·5 50·2 50·0	17·2 9·3 13·8	174·0 160·0 182·7	49·3 48·2 51·0	17·3 17·2 19·5
13·2	16·3	7·0	5·5	6·7	2·5	— 0·2	4·5	22·7	2·8	2·3
123·0 *97·7* *114·2*	*126·7* *102·3* *108·7*	*65·0* *50·8* *58·7*	*29·2* *26·5* *30·4*	*42·2* *29·7* *36·2*	*40·0* *31·0* *34·5*	*48·2* *37·3* *43·3*	*11·3* *7·7* *9·7*	*161·3* *134·6* *151·2*	*51·7* *42·2* *47·8*	*17·7* *15·2* *17·8*
16·5	*16·4*	*7·9*	*1·0*	*6·5*	*3·5*	*6·0*	*2·0*	*16·6*	*5·6*	*2·6*
133·0 115·0 129·8	138·5 116·3 136·3	65·3 57·2 65·2	39·2 31·2 38·0	48·7 37·2 43·7	44·0 37·2 40·0	50·2 44·0 48·8	13·7 10·6 12·8	179·0 155·0 178·5	52·8 47·8 50·8	18·2 15·8 17·8
14·8	20·0	8·0	6·8	6·5	2·8	4·8	2·2	23·5	3·0	2·0
113·7 *107·0* *114·5*	*117·8* *115·6* *122·0*	*59·3* *53·8* *58·2*	*30·7* *30·7* *32·7*	*42·3* *35·8* *39·2*	*39·8* *35·0* *36·2*	*46·5* *40·2* *44·3*	*10·8* *8·7* *11·8*	*156·2* *144·8* *154·3*	*50·2* *47·0* *48·0*	*17·0* *15·0* *17·2*
7·5	*6·4*	*4·4*	*2·0*	*3·4*	*1·2*	*4·1*	*3·1*	*9·5*	*1·0*	*2·2*
126·2 114·7 129·2	130·2 116·3 131·8	69·2 57·8 67·5	37·2 32·8 38·2	50·2 37·8 45·3	40·0 38·0 41·2	49·5 42·7 49·8	10·8 10·7 14·8	181·7 156·2 177·7	51·8 45·0 47·5	18·5 16·3 17·3
14·5	15·5	9·7	5·4	7·5	3·2	7·1	4·1	21·5	2·5	1·0
114·2 *104·3* *115·3*	*123·2* *109·3* *121·0*	*60·2* *51·7* *56·8*	*31·0* *29·0* *31·5*	*46·3* *34·7* *39·0*	*38·2* *34·5* *38·0*	*43·7* *38·2* *42·7*	*18·0* *11·2* *13·7*	*155·0* *141·0* *153·8*	*42·8* *42·0* *44·3*	*17·3* *16·2* *16·3*
11·0	*11·7*	*5·1*	*2·5*	*4·3*	*3·5*	*4·5*	*2·5*	*12·8*	*2·3*	*0·1*
126·3 118·5 131·5	128·7 120·7 135·3	72·0 58·2 65·5	42·0 31·5 36·3	53·2 41·0 48·0	46·3 42·0 45·7	56·7 43·7 48·8	14·3 12·8 14·7	193·0 160·0 177·2	55·2 47·0 51·0	20·2 16·2 17·5
13·0	14·6	7·3	4·8	7·0	3·7	5·1	1·9	17·2	4·0	1·3
118·7 *113·8* *128·8*	*125·0* *122·5* *133·7*	*64·2* *57·3* *63·7*	*33·2* *33·2* *35·3*	*43·8* *39·8* *43·3*	*39·2* *39·2* *42·5*	*48·5* *42·7* *48·2*	*13·2* *7·8* *10·2*	*171·4* *152·3* *164·5*	*47·7* *44·8* *47·0*	*16·0* *15·3* *17·0*
15·0	*11·2*	*6·4*	*2·1*	*3·5*	*3·3*	*6·5*	*2·4*	*12·2*	*2·2*	*1·7*
120·2 115·7 127·3	119·3 117·8 130·5	62·2 57·8 65·5	33·3 30·7 36·8	45·2 39·5 46·5	41·8 38·7 42·2	48·0 43·3 48·8	13·6 10·3 12·8	170·2 155·0 178·0	53·0 46·8 49·0	10·2 16·3 18·3
11·6	12·7	7·7	6·1	7·0	3·5	5·5	2·3	23·0	2·2	2·0
122·2 *112·0* *125·5*	*120·3* *115·8* *128·5*	*66·8* *55·3* *61·8*	*28·8* *26·8* *29·5*	*43·2* *36·5* *41·0*	*42·0* *37·0* *40·2*	*47·8* *41·5* *45·3*	*13·3* *8·2* *10·5*	*167·7* *141·0* *161·3*	*51·2* *44·8* *48·0*	*16·8* *16·2* *17·5*
13·5	*12·7*	*6·5*	*2·7*	*4·5*	*3·2*	*3·8*	*2·3*	*20·3*	*3·2*	*1·3*
125·5 113·7 126·5	128·8 117·5 130·2	67·8 59·0 67·2	47·2 35·2 42·5	51·7 41·5 50·0	37·7 37·5 43·2	44·2 41·0 48·8	13·7 10·7 12·2	190·5 161·0 180·8	50·0 45·8 48·3	18·5 16·0 18·0
12·8	12·7	8·2	7·3	8·5	5·7	4·8	1·5	19·8	2·5	2·0
120·0 *115·2* *128·3*	*120·8* *117·0* *131·8*	*64·8* *58·2* *65·3*	*33·2* *34·2* *34·3*	*38·3* *36·8* *42·0*	*38·0* *37·5* *41·8*	*49·2* *43·0* *46·8*	*11·8* *8·3* *10·5*	*167·5* *157·5* *172·0*	*54·0* *46·7* *51·7*	*17·7* *16·3* *19·0*
13·1	*14·8*	*7·1*	*0·1*	*5·2*	*4·3*	*3·8*	*2·2*	*14·5*	*5·0*	*2·7*
121·0 112·2 124·3	125·8 117·2 132·3	65·7 58·5 64·8	42·2 35·7 42·2	48·2 40·2 47·5	42·2 38·7 44·2	53·0 43·8 49·3	11·2 10·0 14·7	177·7 161·2 183·5	53·0 46·7 49·3	17·2 16·7 18·5
12·1	15·1	6·3	6·5	7·3	5·5	5·5	4·7	22·3	2·6	1·8

As stated before, the control groups in the Slaughter Test Experiment consisted of only two tollies of each herd. Although the figures are given for these control groups, the smallness of the number of animals precludes any deductions being drawn from them.

(*d*) The Influence of Bonemeal Feeding on the Quantity and Quality of Milk.

As yet no figures are available with regard to this question. The half-bred batch I heifers of the Fries and Red Poll herds are calving as this report is being written and accurate records will be kept of the milk produced by them.

(*e*) The Influence of Bonemeal Feeding on Skeletal Development.

To determine the influence of bonemeal feeding upon skeletal development three bonemeal and three control basis herd cows were selected from each herd in March, 1927. With the "Deriaz" measuring stick (see Figures 69 and 70) the following body measurements were taken:—

1. *Length of body.*—The horizontal distance between the point of the shoulder and the pinbone.
2. *Height of body.*—The vertical distance from the ground to the top of the withers (Fig. 69).
3. *Height at hook bones.*—The vertical distance from the ground to the base of the sacrum.
4. *Depth of chest.*—Vertical distance between vertebral column and sternum, taken just behind the shoulder (Fig. 70).
5. *Width of chest.*—Taken just behind the shoulders.
6. *Width between the hook bones.*
7. *Width between the thirls.*
8. *Length of croup.*—The horizontal distance from hook bone to pinbone.
9. *Width between pinbones.*
10. *Heart girth.*—Taken with a steel measuring tape directly behind the shoulder.
11. *Length of Head.*—The distance from the poll to the end of the nasal bones.
12. *Width between the Eyes.*—The horizontal distance between the inner commissures.

It will be noticed that, wherever possible, the measurements were taken between skeletal projections which are not covered with flesh or fat. This was done to minimize experimental error in measuring.

To minimize the experimental error further, the animals were placed on a level concrete floor, with their legs placed naturally and symmetrically under their bodies. After the first set of readings had been taken the animal was walked round, again placed in position, and a second set of readings taken. A third set of readings was obtained in the same way and for recording purposes, the average of the three readings was then calculated.

The bulls were also measured and, in addition, the twelve to fifteen months old calves of the measured cows.

As far as possible cows with heifer calves were selected, as it was the idea to obtain body measurements of bonemeal and control *families* of cattle up to the fourth generation.

From the first to the 17th of March, 1927, three bonemeal and three control basis herd cows of each herd, three bonemeal, and three control half-bred batch I calves of each herd and two bulls of each herd were measured; in all 54 animals. The next year (10.3.1928) the surviving cows and calves of the original lot were again measured, as also all the half-bred batch II calves of the original cows, and the new herd bulls. The old herd bulls had, in the meanwhile, been disposed of.

We have, therefore, at present two sets of readings for the basis herd cows and batch I of the half-breds, and one set of readings of batch II of the half-breds.

In table No. 20 the measurements of the bonemeal and control cows of each herd are recorded. Under the readings for each cow the two sets of readings of their first calves are given; the first set was taken when the calf was 12 to 15 months old, and the second set a year later. In this way it is easy to compare the skeletal development of the calf with that of its dam. All measurements are given in centimeters and the weights in pounds.

Comments on these figures are as follows:—

(*a*) The first point of interest in studying table No. 20 is that, with one exception (cow 1234), the control basis herd cows show smaller body measurements than do the bonemeal basis herd cows. The average measurements for bonemeal and control basis herd cows are given in table No. 21.

TABLE No. 21.

	Length of body.	Height at withers.	Height at hook-bones.	Depth of chest.	Width of chest.	Width between hookbones.	Width between thirls.	Length of croup.	Width between pinbones.	Heart girth.	Length of head.	Width between eyes.
Bonemeal cows	147·7	124·1	127·6	66·8	39·7	49·3	41·9	49·6	13·4	180·8	52·2	17·1
Control cows...	*139·9*	*118·5*	*122·3*	*63·1*	*31·0*	*42·7*	*39·5*	*47·3*	*13·1*	*163·2*	*49·6*	*17·1*
Difference.....	7·8	5·6	5·3	3·4	8·7	6·6	2·4	2·3	0·3	17·6	2·6	0·0

It will be noticed that there is a marked difference between bonemeal and control basis herd cows in length of body, width of chest, heart girth, and width between hookbones; while there is a little difference between them in regard to depth of chest and height, either at the withers or at the hookbones.

From the above figures it is possible to picture the control cows as leggy and undeep and narrow of body; while the bonemeal cows are set lower to the ground and show better depth and width of body.

(*b*) From table No. 20 it will be seen that, of the seven bonemeal-fed calves, five were equal to their dams in skeletal development at the age of two years and three months, and that the body measurements of the remaining two are but very little below those of their mothers. In a few instances the maternal readings are higher than those of the calves, but it will be noticed that this occurs especially under the headings: width between hookbones, width between pinbones and, to a lesser extent, under the heading: width between thirls. It may, however, be presumed that these greater measurements of the cows are due to their having calved while the youngsters had not.

(*c*) Although in body height, both at the withers and at the hookbones, most of the control calves are equal to or even surpass their dams, in the other measurements they are deficient, especially in depth of chest.

(*d*) Table No. 22 gives the average body measurements for bonemeal and control calves, both at the ages of one year and three months, and two years and three months, respectively.

TABLE NO. 22.

	Body weight.	Length of body.	Height at withers.	Height at hookbones.	Depth of chest.	Width of chest.	Width between hookbones.	Width between thirls.	Length of croup.	Width between pinbones.	Heart girth.	Length of head.	Width between eyes.
Bonemeal calves, 1927	664·6	128·0	115·3	117·7	58·4	32·7	39·7	38·4	44·5	10·6	158·3	46·8	16·4
Control calves, 1927......	*537·5*	*120·3*	*108·3*	*113·8*	*54·5*	*30·1*	*35·6*	*35·7*	*40·5*	*8·6*	*145·2*	*44·6*	*15·7*
Difference between bonemeal and control calves, 1927	127·1	7·7	7·0	3·9	3·9	2·6	4·1	2·7	4·0	2·0	13·1	2·2	0·7
Bonemeal calves, 1928	954	141·0	128·4	132·9	66·1	38·8	47·0	42·2	49·2	13·9	179·8	49·6	18·1
Control calves, 1928......	*700*	*132·9*	*121·1*	*126·0*	*60·8*	*32·3*	*40·1*	*38·9*	*45·1*	*11·1*	*159·5*	*47·8*	*17·5*
Difference between bonemeal and control calves, 1928	254	8·1	7·3	6·9	5·3	6·5	6·9	3·3	4·1	2·8	20·3	1·8	0·6

The above table appears to furnish conclusive proof that the skeletons of bonemeal-fed animals develop faster and become bigger than do skeletons of animals receiving no bonemeal. It will be noticed that in 1927 the bonemeal calves were superior in every respect to the control calves and that this superiority is not only maintained in 1928, but is increased for every measurement.

(*e*) The tendency of skeletal development and growth is not the same for bonemeal and control animals. This will be seen from table No. 23, which gives the percentage of increase in each of the 12 measurements, for both bonemeal and control groups, over the period 17.3.1927 to 10.3.1928.

TABLE NO. 23.

	Percentage Increase 17.3.1927 to 20.3.1928.												
	Body weight.	Length of body.	Height at withers.	Height at hookbones.	Depth of chest.	Width of chest.	Width between hookbones.	Width between thirls.	Length of croup.	Width between pinbones.	Heart girth.	Length of head.	Width between eyes.
Half-bred batch I bonemeal.......	43·5	10·2	11·4	12·9	13·2	18·7	18·4	9·9	10·6	31·1	13·6	5·9	10·4
Half-bred batch I control.....	*30·2*	*10·5*	*11·8*	*10·7*	*11·6*	*7·3*	*12·6*	*9·0*	*11·4*	*29·1*	*9·8*	*7·2*	*11·5*

It will be seen, from the above table, that the bonemeal animals show a more marked development in depth of chest, width between hookbones, thirls, and pinbones, and in heart girth, than do the control animals. On the other hand, the latter show a definite tendency to be higher on the front legs, longer of head and croup, narrower of chest and smaller of heart girth.

The figures in table No. 23 seem to bear out the statement, made earlier in this article, that " on phosphorus deficient pastures, the body-build is liable to become off type." The animals show a tendency to become longer of leg, narrower and less deep of chest, narrower over hookbones, thirls, and pinbones, longer of croup and head, and smaller of heart girth.

The figures in table No. 23 further tend to show that the correction of the phosphorus deficiency by bonemeal feeding tends to prevent the body formation from becoming " off type," in that such a correction seems to produce depth and width of body, to prevent legginess and to keep the animals nearer to the ground.

The investigational work with regard to skeletal measurements will, of course, be continued for the whole period of the main experiment. So far, only measurements of the live animals have been taken, but a representative number of bonemeal and control animals of each generation will be slaughtered, in order to obtain comparative data in regard to the measurements, weights, and composition of the individual bones.

(*f*) THE INFLUENCE OF BONEMEAL ON OSTEOPHAGIA.

Graphs Nos. 11, 12, 13, and 14 show curves representing the craving for rotten bones in the bonemeal and control half-bred batch I animals of each of the four herds.

As explained earlier in this paper, all experimental animals are tested for " pica " every 14 days and are classified, according to the degree of craving they show, into:—

(*a*) Rotten bone cravers.
(*b*) Sweet bone cravers.
(*c*) Non-cravers.

The rotten bone cravers are, of course, the worst cravers and show the most pronounced mineral deficiency. Hence the influence of bonemeal feeding is best demonstrated by its effect on rotten bone craving, which is indicated in the individual herd graphs (Nos. 11, 12, 13, and 14).

A study of these graphs reveals some interesting points:—

(*a*) It will be seen that the control curve of each herd goes higher and higher, as the animals increase in age and weight, while the bonemeal curves, after an initial rise, come down to a minimum. Bonemeal supplies the mineral requirements of the body for phosphates and removes craving.

(*b*) The curves start in October, 1926, with eight months old calves and end in August, 1928, with 30 months old animals. In November, 1926, the calves were weaned and all four graphs show a definite increase in craving of both the bonemeal and control groups directly after weaning. This temporary increase may be ascribed to the habituation to new nutritional conditions, rendered necessary by the change from mother's milk to pasture only. The initial tendency in all the curves is in an upward direction, but whereas the bonemeal group curves come down, and remain low, the control curves continue to rise to a high level, and remain high.

(*c*) It is, of course, quite impossible at this stage of the investigations to form any definite opinion as to the influence of improved blood upon osteophagia. As yet only half-bred animals are available and even the oldest of these are only 2½ years old. Hence, the available data are quite insufficient to allow us to draw final conclusions. Nevertheless, these data, taken together with the results of close observation of the animals themselves, enable us to point to some apparent tendencies in the half-breds of the different herds and to indicate possible contributing factors which affect these tendencies:—

(1) It will be noticed that the increase in craving directly after weaning is most marked in the Fries herd, less so in the Africander and Sussex, and least in the Red Poll. This is true both for the bonemeal and control groups. Among these four breeds, Fries blood is, without doubt, the most highly specialized and a marked reaction to change of conditions, as shown in the Fries osteophagia curves, was to be expected.

The sudden rise of the Fries control curve, and the continued high degree of craving would tend to indicate that, of the four herds, the Fries half-breds are the most sensitive to phosphorus deficiency.

On the other hand, the prolonged period of habituation, as shown by the pica curve of the Fries bonemeal-fed half-bred calves, would tend to show either that the Fries half-breds:

(*a*) need more bonemeal than the half-breds of the other herds, or

(*b*) are unable to utilize the phosphorus supplied as economically as the other breeds.

(2) Judging from the Africander control curve, it would appear as if half-breds of this breed are second only to Fries half-breds in their sensitiveness to a phosphorus deficiency.

(3) The Sussex and Red Poll half-breds appear to withstand phosphorus deficiency better than either Fries or Africander half-breds during the period when they are eight months to 16 months of age. The control curves of the Sussex and Red Polls show a prolonged, low degree of craving.

(4) The Sussex and Red Poll bonemeal curves drop to zero or thereabouts very soon after weaning; this would seem to indicate that the grades of these breeds either—

(*a*) need less phosphorus, or

(*b*) make more economical use of the phosphates supplied to them than do the Africander and Fries half-breds.

(5) Of the Sussex and Red Poll half-breds the latter appear to be less sensitive to phosphorus deficiency and able to make better use of its bonemeal than the former.

C. THE EFFECT OF BONEMEAL FEEDING ON THE INCIDENCE OF DISEASE.

Under this heading the basis herd cows and the half-bred calves will be dealt with separately. In this article mortality figures only are given; however, a new system of recording has now been introduced which will allow all the details of the diseased conditions from which the experimental cattle may suffer to be recorded. In this way it is hoped to be able to study the effect of bonemeal feeding on the incidence of disease in greater detail.

(1) The Basis Herd Cows.

Going back to table No. 1 it will be seen that, during the period 18.3.1925 to 30.6.1928, 14 bonemeal cows died out of a total of 157. This works out at 8.9 per cent. in three years and three months, or about 2.7 per cent. per year. Of the 92 control cows 61 died during the same period. This works out at 66.3 per cent. in three and a quarter years, or 20.4 per cent. per year. These figures are only of interest if studied in conjuction with a table giving the various causes of death, and the number of animals which died from each of these causes. In table No. 1 the details with regard to causes of death are given. In table No. 24 the number of deaths, due to each cause, has been worked out as percentages of the total number of cows in the experiment and also as percentages of the total number of deaths.

TABLE NO. 24.

	Lamsiekte.	Poverty.	Vegetable Poisoning.	Fractures.	Quarter-evil.	Tuberculosis.	Arsenical Poisoning.	Accidental Death.	Pneumonia.	Unknown Causes.
Number of bonemeal cows dead............	3	0	3	2	1	1	1	1	1	1
Percentage deaths to total number of bonemeal cows in the experiment...........	1·9	0·0	1·9	1·3	0·6	0·6	0·6	0·6	0·6	0·6
Percentage of total number of deaths.........	21·4	0·0	21·4	14·3	7·1	7·1	7·1	7·1	7·1	7·1
Number of control cows dead.................	*40*	*6*	*5*	*5*	*1*	*0*	*3*	*0*	*0*	*1*
Percentage control deaths to total number control cows in the experiment................	*43·5*	*6·5*	*5·4*	*5·4*	*1·1*	*0·0*	*3·3*	*0·0*	*0·0*	*1·1*
Percentage of total number of deaths.............	*65·6*	*9·8*	*8·2*	*8·2*	*1·6*	*0·0*	*4·9*	*0·0*	*0·0*	*1·6*

(*a*) *Lamsiekte.*

From table No. 24 it may be seen that lamsiekte is responsible for the greatest number of deaths, but, whereas in the control herd 40 out of the total number of 92 cows died of lamsiekte (i.e. 43.5 per cent.) only three bonemeal cows or 1.9 per cent. died of this disease.

Going into the history of the three bonemeal cows which died of lamsiekte, it was found that two of these cows were chronic cravers, i.e. animals which, although persumably no longer in need of phosphorus, retained a perverted appetite for putrid material. Such cases are not uncommon amongst native stock bred in the area.

The influence of bonemeal feeding on the incidence of lamsiekte is, therefore, very pronounced.

(*b*) *Poverty.*

It will be noticed that six animals or 6.5 per cent. of the total number of control cows died of poverty, whereas no bonemeal animals died of this cause. The records show that deaths due to poverty occur mostly during the late winter, when the pasture is at its worst and the drain on the system of the cow, due to lactation, is greatest. In fact, during the months of September and October, most of the control cows are so weak that they cannot go out with the rest of the cattle to graze in the camps, but have to be kept in paddocks near the homestead. It is very common to have to lift the control cows in the morning during this time of the year, the animals being too weak to rise by themselves. Such bodily weakness has never been observed in the bonemeal cows. They, of course, also fall off in condition, but except in the case of very old animals, are never reduced to a state of great weakness or emaciation.

(c) *Vegetable and Arsenical Poisoning.*

These causes were responsible for the deaths of eight controls (8.7 per cent.) and four bonemeal cows (2.5 per cent.). The controls, due to the mineral deficiency, are continually searching for the necessary substances with which to satisfy their craving. They roam far more than do the bonemeal animals and eat anything which has an abnormal taste. It is evident that under these conditions poisoning is very apt to occur amongst the controls.

(d) *Fractures.*

Here again the controls show 5.4 per cent. mortality, as compared with 1.3 per cent. mortality amongst the bonemeal cows. The cases of fractures amongst the controls were due to slipping, injuries by other animals, etc., i.e. the causes which are likely to produce fractures only in weak or brittle-boned animals.

Of the two bonemeal cows which had to be destroyed on account of fractures, one accidentally put her hind foot in a hole in the dolomite formation and was unable to withdraw it. When she was found next morning with the foot in the hole, the femur and tibia of the off hind leg were both badly fractured. The second case was of a similar nature.

The main causes of death are, therefore, in the order of their importance:—Lamsiekte, poverty and poisoning. In all these cases the control animals are particularly susceptible as a direct result of the mineral deficiency. The influence of bonemeal feeding is very well demonstrated by the figures quoted above.

(2) THE HALF-BRED BONEMEAL AND CONTROL CALVES.

Table No. 2 shows that, out of the total of 344 bonemeal and 81 control calves born alive, 13 (3.8 per cent.) bonemeal calves and 18 (22.2 per cent.) control calves died during the period 1/1/1926 to 30/6/1928. This works out at 1.5 per cent. (bonemeal) and 8.9 per cent. (control) per year. Table No. 25 gives the same details for the half-bred calves as table No. 24 gave for the basis herd cows.

Table No. 25 shows that Lamsiekte is also the cause of the greatest number of deaths amongst the half-bred animals. It has, in 2½ years' time, accounted for 8.6 per cent. of the total number of half-bred controls (i.e. 38.9 per cent. of total deaths among the control calves). Vegetable poisoning and fractures here too come next in seriousness, each being responsible for the death of 3.7 per cent. of the half-bred controls (16.7 per cent. of total deaths). It is noticeable, that no bonemeal animals died of any of the above causes.

Seven bonemeal and two control calves died of quarter-evil in March, 1927, on the farm Biesjiesvlakte. As this farm is situated

13½ miles away from headquarters, and carcases have to be transported that distance for post-mortem examination, there was some delay in diagnosing the outbreak and inoculating the calves with a preventive vaccine. Had it not been for this outbreak of quarter-evil, which, of course, has no bearing on the experiment as such, the comparison between bonemeal and control calves would have been still more in favour of the bonemeal group. If we omit the nine deaths from quarter-evil, the total number of deaths among the bonemeal group would be six (or 1.7 per cent.) and among the control group 16 (or 19.75 per cent.). Calculated in this way, the death rate among the control calves is more than 11 times as high as among the bonemeal-fed animals.

TABLE No. 25.

	Lamsiekte.	Vegetable Poisoning.	Quarter-evil.	Fractures.	Sweating Sickness.	Traumatic Pericarditis.	Arsenical Poisoning.	Accidents.	Pneumonia.	Unknown Causes.
Number of bonemeal half-bred animals which died..................	0	0	7	0	1	0	1	1	1	2
Percentage deaths to total number of calves in experiment........	0·0	0·0	2·0	0·0	0·3	0·0	0·3	0·3	0·3	0·6
Percentage of total deaths	0·0	0·0	53·8	0·0	7·8	0·0	7·8	7·8	7·8	15·4
Number of control half-bred animals which died	*7*	*3*	*2*	*3*	*0*	*1*	*0*	*0*	*0*	*2*
Percentage of deaths to number of calves in experiment...........	*8·6*	*3·7*	*2·5*	*3·7*	*0·0*	*1·2*	*0·0*	*0·0*	*0·0*	*2·5*
Percentage of total deaths	*38·9*	*16·7*	*11·1*	*16·7*	*0 0*	*5·6*	*0·0*	*0·0*	*0·0*	*11·1*

The significance of the figures quoted in this section can scarcely be over-estimated. In the brief communication of Theiler, Green, and Du Toit (1928) on the preliminary results of these investigations, the statement was made that: "the cows limited to the natural grazing are *dying out*, and if the same mortality incidence continues one branch of the experimental programme will disappear. The farm will then have been shown to be *worthless for cattle rearing without mineral supplement*."

This prediction has been proved to be true in the course of the investigations.

The experiment started with 65 control cows, but the mortality was so high that another 27 had to be added during the three years to keep the control groups from disappearing altogether. Actually 61 cows died among the 92 controls, whereas only 14 died out of 157 bonemeal-fed cows (for detailed figures see table No. 1).

But not only did this high mortality occur among the control cows; table No. 2 shows that their calves—already small in number owing to the low fertility of the cows—suffered the same fate.

The first year of the experiment 37 calves were born in the control groups; the next year 28, and the third year 16. These figures show clearly how the herd gradually faded out. Among these calves the mortality, as was shown above, was many times as high as among their bonemeal-fed contemporaries.

If we consider these factors:—

(1) *Very high mortality among the cows*,
(2) *very low fertility of cows*,
(3) *high mortality among their calves*, and (what must undoubtedly be expected, when these calves have grown up and have to reproduce their kind again),
(4) *the same very high mortality among the next generation of cows, and so forth*,

we are forced to the conclusion that the above statement is no exaggeration, but that, probably within two or three generations, the control herd will have *died out* completely and literally, unless the herd is continually being replenished.

There can be no question that, under such conditions cattle farming becomes impossible; and we may indeed conclude that such a farm is "*worthless for cattle rearing.*"

A study of the data quoted in this paper shows how completely the picture is changed when the cattle receive their mineral ration. The cows remain in good condition, the mortality amongst them is reduced to a negligible figure, the fertility and "calf crop" is high, the calves remain healthy and in good condition, and a normal increase in the herd is assured.

VII. SUMMARY AND CONCLUSIONS.

1. The present investigations form a continuation of the investigations into *Lamsiekte* (or *Parabotulism*) of cattle, started about 20 years ago by officers of this Division.

The solution to the lamsiekte problem was found about 10 years ago when it was shown that the primary factor which led to this disease was *phosphorus deficiency of the soil and pasture*, which produced an abnormal craving for bones and carrion in cattle grazing on such lands. If this carcase debris is infected with a specific toxicogenic organism (*Clostridium parabotulinum bovis*) lamsiekte is produced.

2. It was found, in the course of the lamsiekte investigations, that the disease could be prevented by supplying phosphorus, in the form of *bonemeal*, to the animals.

Further investigations proved that bonemeal not only prevented lamsiekte, when given in small daily rations to cattle, but was also responsible for a most remarkable *rate of growth* and *improvement in condition*. Further advantages of bonemeal feeding were observed and will be referred to again.

3. The investigations just mentioned had solved the problem of cattle breeding on phosphorus deficient veld, in so far as the ordinary *native stock* was concerned.

The next step was to determine whether it was possible to *improve the stock* by grading up the native cattle with pure bred bulls, provided the animals received the necessary ration of bonemeal.

4. Bonemeal was found to have a remarkable influence on the *fertility of the cows.* It was found, for instance, that of the 109 " bonemeal cows " which remained in the experiment for 3 years, 66.1 per cent. had three calves, whereas of the 20 " control cows " (receiving no bonemeal) which were in the experiment for the same period, not one had three calves.

During the first three years of the experiment the bonemeal cows produced 87.3 per cent. of the possible number of calves, whereas the control cows only produced 56.5 per cent. of the possible number.

Other interesting figures are given in the body of the report.

5. It would seem that cows grazing on a deficient pasture exhaust their mineral reserves in the process of gestation, and generally need two seasons or more to build up their reserves again before they will become pregnant once more.

If bonemeal is fed to such cows they will breed with much greater regularity.

6. Although the investigations have not proceeded for a sufficiently long time to enable us to quote large numbers, it would appear as if calves born of mothers receiving bonemeal, and themselves receiving bonemeal since the age of weaning, attain *sexual maturity* earlier than the " control " calves of " control " mothers.

In the experiment 84.4 per cent. of the bonemeal heifers became pregnant when put to the bull at the age of 2 to 2¼ years, whereas only 73.7 per cent. of the control heifers became pregnant.

7. Cattle receiving bonemeal seem to retain their *constitutional vigour* far better than do the control cattle which receive no mineral ration.

8. In regard to the *sex* of the grade calves, it was found that in the Fries, Red Poll and Sussex herds there was a slight preponderance of males over females, whereas in the Africander herd there were 36 bull calves to 48 heifers.

Further observations will show whether any significance has to be attached to this difference.

9. There is no appreciable difference in the *weight at birth* between the calves of bonemeal cows and those born of cows receiving no bonemeal. The calves in the Africander herd weighed about 6 per cent. less at birth than the calves in the other three herds.

10. At the *age of weaning* (about nine months) the calves of bonemeal mothers weigh about 16.5 per cent. more than the calves of control cows.

This difference is due, not to the quality of the milk (which is the same for bonemeal-fed and control cows), but to the larger milk yield of the bonemeal cows.

11. The influence of bonemeal on the *rate of growth* of calves is very remarkable. At birth there is no difference in weight between

"bonemeal" calves and "control" calves; at six months there is a difference of 48 lb. (or 14.5 per cent.) in favour of the former group; and at 2½ years the difference is 243 lb. (or 33.2 per cent.).

It was found that this additional weight at 2½ years was obtained at a cost (for bonemeal) of 8s. 9d., or ½d. per pound live weight.

12. A comparison of the rate of growth of all grade calves, between the season 1926/1927 and 1927/1928 reveals some interesting differences. The influence of continuous or discontinuous rainfall on the pasture, and the resultant influence on the winter condition of the animals, is discussed in some detail.

13. Comparing the grades of the different breeds it was found that the *Sussex grades* are heaviest at six months and remain heavier than the other groups; at 2½ years the bonemeal-fed animals weigh 1,028 lb. In two years the increase in weight was 152 per cent.

The *Red Poll grades* receiving bonemeal weigh considerably less than the other groups at six months, but overtake the Africander and Fries groups. The rate of growth is remarkable: in two years the increase in weight is 198 per cent.

The *Fries* "bonemeal" grades increase 144 per cent. in two years, and the *Africander* grades show about the same rate of increase.

14. The rate of growth in the "*control groups*" (receiving no bonemeal) of these herds differs considerably in the four breeds. In the Africander grades it is 99 per cent., in the Fries 119 per cent., in the Red Poll 141 per cent., and in the Sussex 132 per cent.

The Red Poll control grades did very well when compared with some of the other controls. The calves of this breed seem to possess a remarkable "growth impetus."

15. Experiments are in progress which are intended to demonstrate the difference in the *quantity and quality of beef* between bonemeal-fed tollies and those receiving no bonemeal. The results are not yet available.

16. It is also hoped to be able, in a future report, to give accurate comparative figures in regard to the influence of bonemeal feeding on the *quantity and quality of milk*. The composition of the milk both for butterfat and for solids-not-fat will be given.

17. The influence of bonemeal feeding on the *skeletal development* of cattle has also been studied. There is a marked difference in favour of cows receiving bonemeal in respect of length of body, width of chest, heart girth and width between hookbones.

The skeletal development of bonemeal-fed calves is also superior in every way to that of the control calves.

18. The effect of bonemeal in reducing *osteophagia* ("pica" or "craving for bones") has been dealt with fully in earlier publications.

In the present article the behaviour of the breeds used in this investigation has been recorded for the period of observation. The *Fries* grades seem to respond most readily to bonemeal feeding; next come the *Africander* grades and then the *Sussex* and *Red Poll*.

19. Remarkable results are recorded relating to the effect of bonemeal feeding on the *incidence of disease*. In the bonemeal-fed herd of cows the mortality was very low, only 14 cows dying in 3¼ years out of a total of 157 (8.9 per cent.). On the other hand, in the control

herd 61 cows died out of 92 (66.3 per cent.). The mortality was so heavy among the controls that their numbers had to be replenished continually.

20. The chief *causes of death* were lamsiekte, poverty, plant poisoning, arsenical poisoning and fractures.

Of 43 cows which died of *lamsiekte* 40 were controls and three bonemeal-fed cows; of six which died of *poverty* all were controls; of the 12 which died of *poisoning* eight were controls; and of the seven cases of *fracture* five were amongst the controls.

21. The same remarkable differences were observed between *bonemeal-fed calves* and *control calves.* Amongst the former the mortality was 3.8 per cent., amongst the latter 22.2 per cent.

22. The *causes of death* were similar to those mentioned above. *Lamsiekte* accounted for seven calves, all of which were controls; of the four calves which died of *poisoning* three were controls; three calves, all controls, died on account of fractures.

Unfortunately, there was an outbreak of quarter-evil amongst these calves, which was responsible for several deaths chiefly among the bonemeal-fed animals. Had it not been for this outbreak, which, of course, has no bearing on the experiment, the figures in favour of the bonemeal-fed calves would have been still more striking.

23. The statement made in a previous publication, that cattle grazing on phosphorus deficient veld would *die out* unless a supplementary mineral ration is supplied, is fully borne out by the results of the present investigation.

On the other hand, the results so far obtained prove that *grading up of native stock on phosphorus deficient veld can be carried out entirely satisfactorily and very economically, provided a small daily ration of bonemeal is supplied.*

LITERATURE.

References to all important articles dealing with Mineral Deficiency in Pastures and its Relation to Animal Nutrition, will be found in Orr's publication which is quoted below. Only those further articles which are discussed in detail in the foregoing paper are quoted here.

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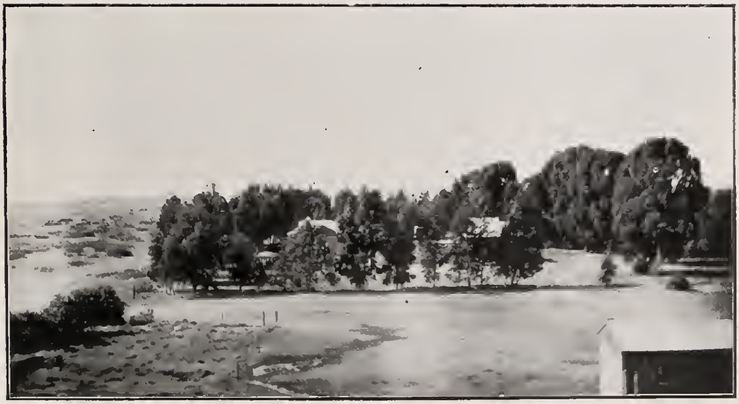
FIG. 1.



Bird's Eye View of Homestead at Armoedsvlakte.

FIG. 2.

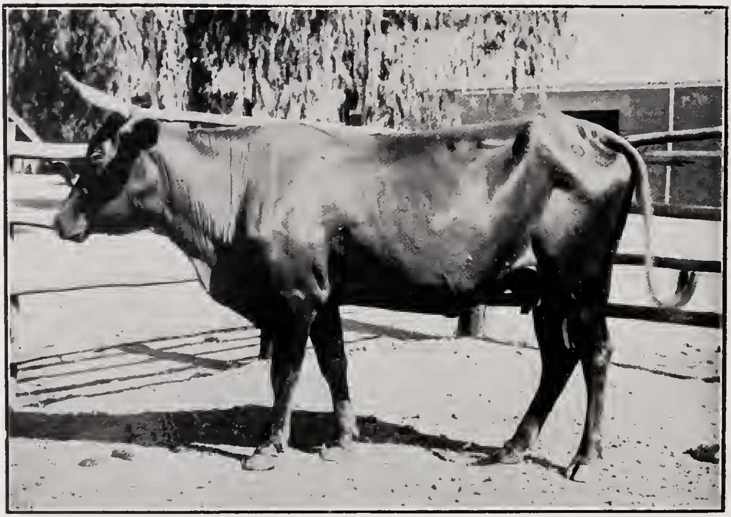
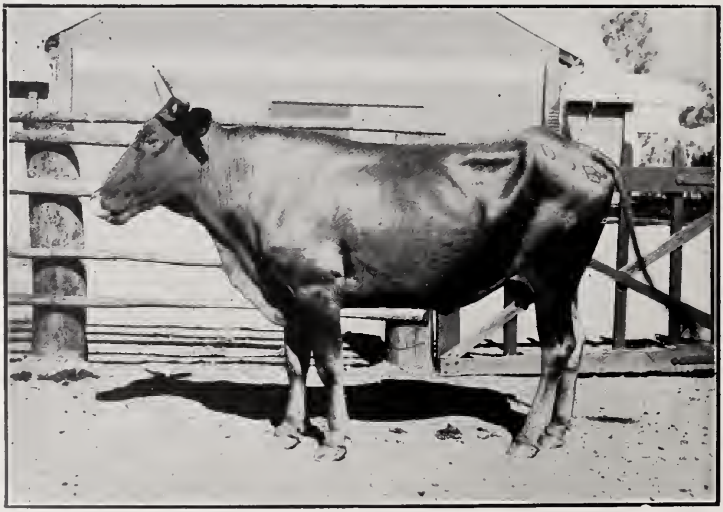


FIG. 3.



FIGS. 2 and 3.—Types of Bone-meal Basis Herd Cows.

FIG. 4.

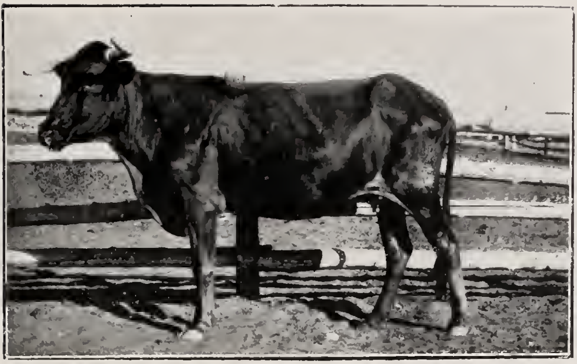


FIG. 5.

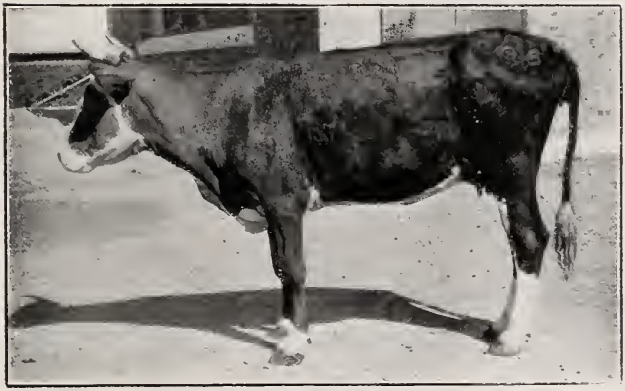
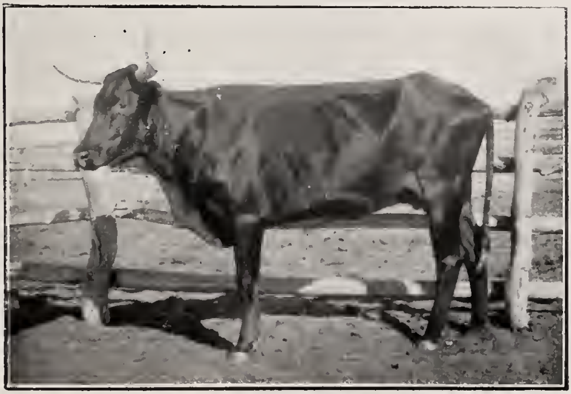


FIG. 6.



FIGS. 4, 5 and 6.—Types of Control Basis Herd Cows in Summer Condition.

Fig. 7.

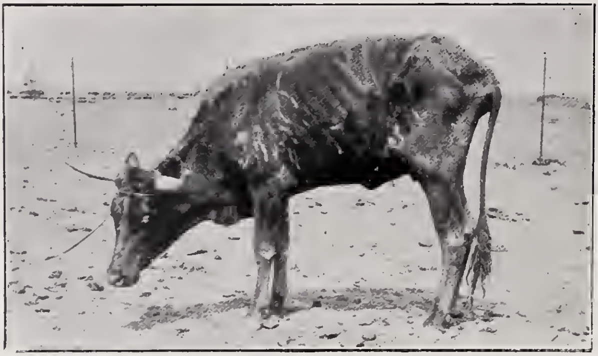


Fig. 8.

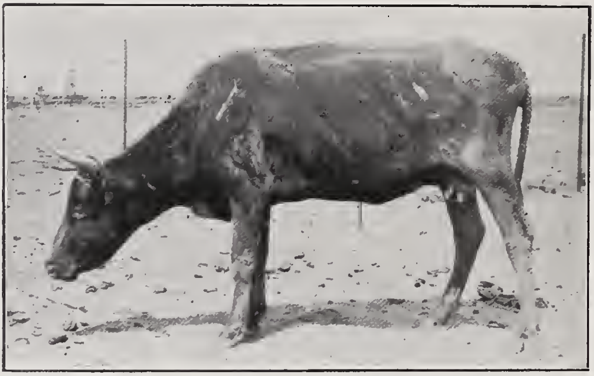
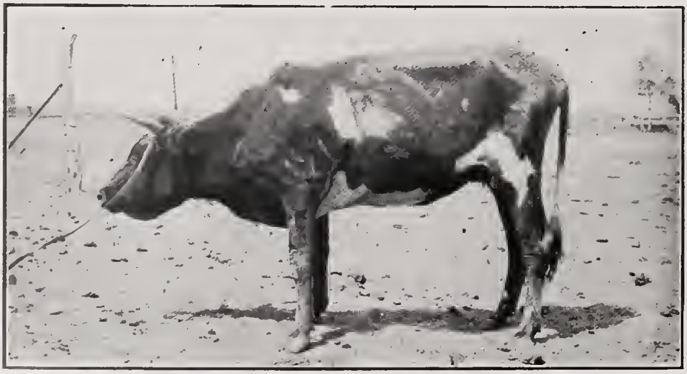


Fig. 9.



Figs. 7, 8 and 9.—Types of Control Basis Herd Cows at the End of Winter.

FIG. 10.

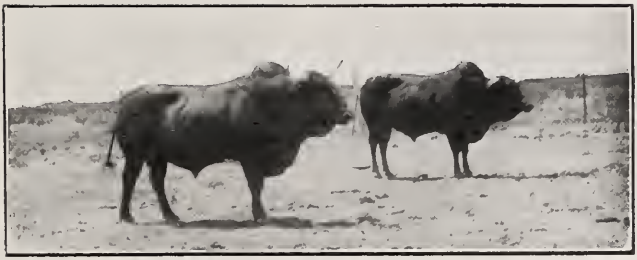
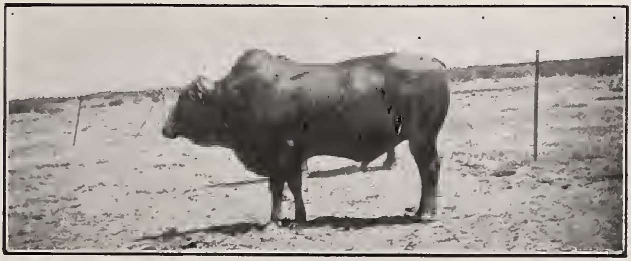


FIG. 11.



FIGS. 10 and 11.—Africander Bulls.

Sires of the Half-bred Grade.

Fig. 12.

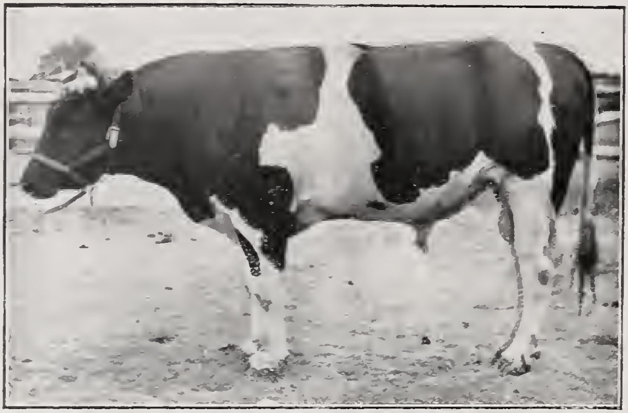
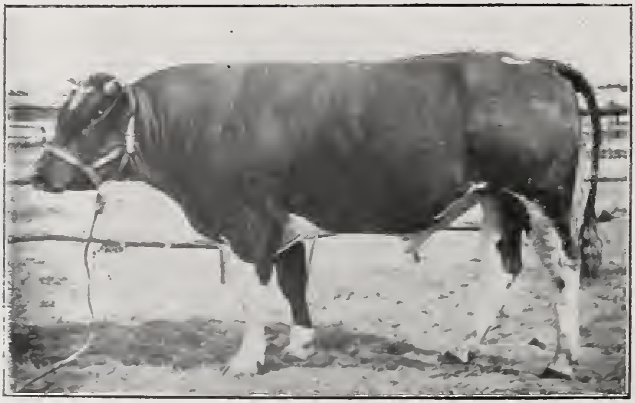


Fig. 13.



Figs. 12 and 13.—Fries Bulls.
Sires of the Half-bred Grade.

FIG. 14.

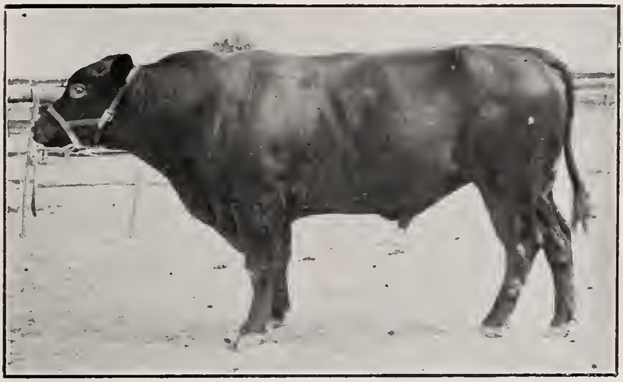
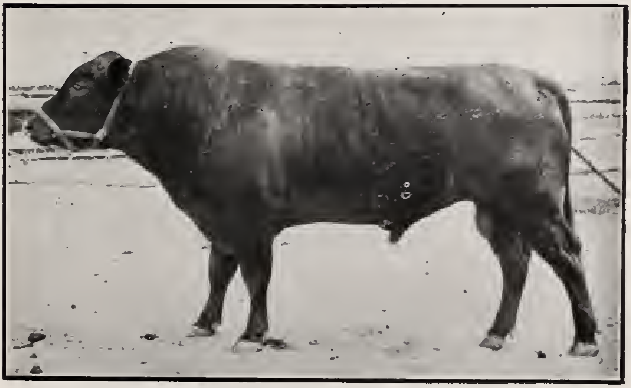


FIG. 15.



FIGS. 14 and 15.—Red Poll Bulls.

Sires of the Half-bred Grade.

FIG. 16.

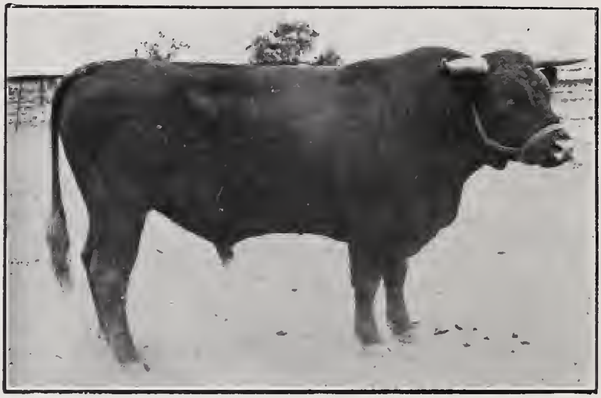
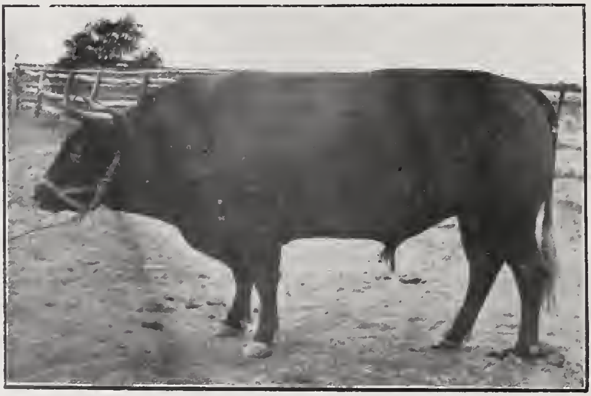


FIG. 17.



FIGS. 16 and 17.—Sussex Bulls.

Sires of the Half-bred Grade.

Fig. 18.

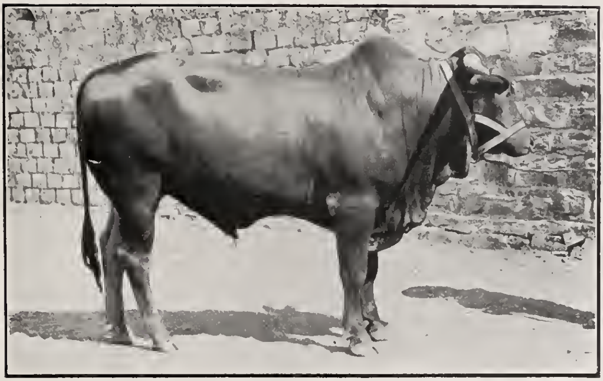
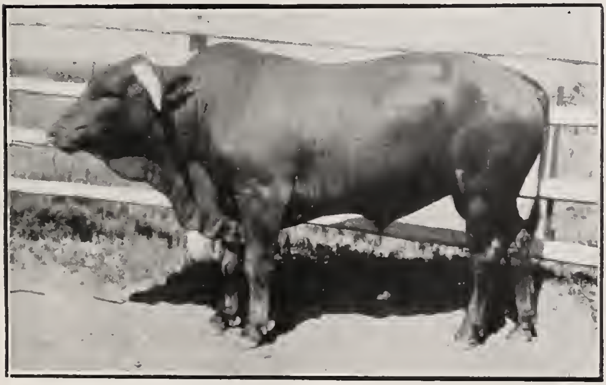


Fig. 19.



Figs. 18 and 19.—Africander Bulls.
Sires of the Three-quarter-bred Grade.

Fig. 20.

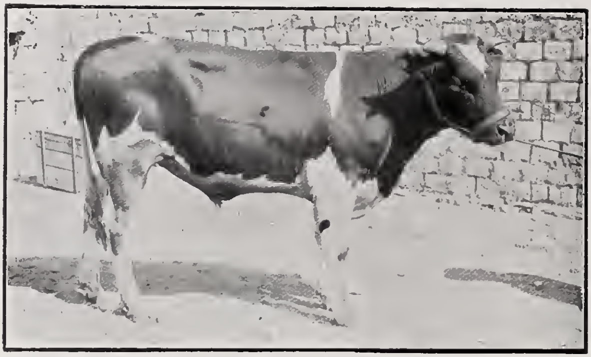
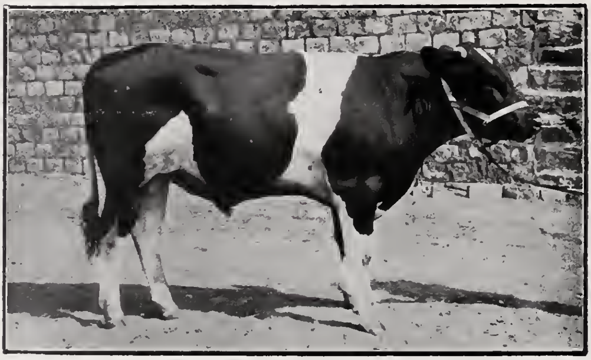


Fig. 21.



Figs. 20 and 21.—Fries Bulls.
Sires of the Three-quarter-bred Grade.

Fig. 22.

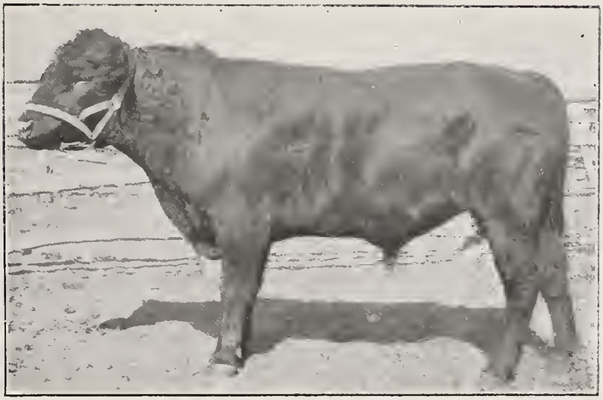
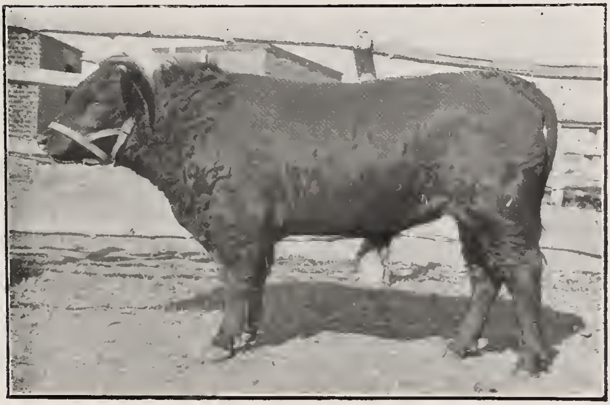


Fig. 23.



Figs. 22 and 23.—Red Poll Bulls.
Sires of the Three-quarter-bred Grade.

Fig. 24.

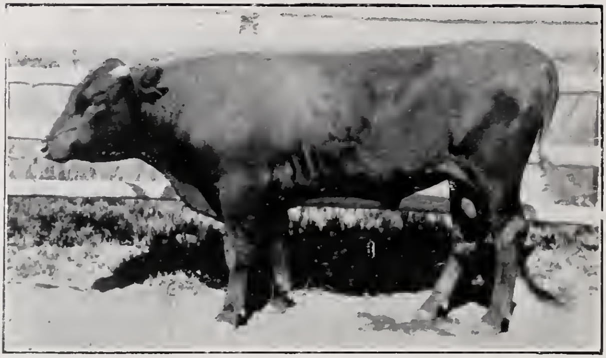
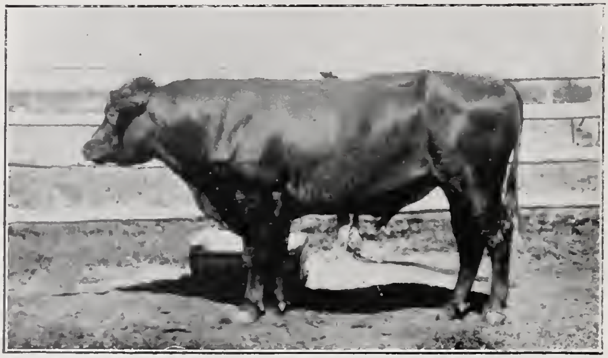
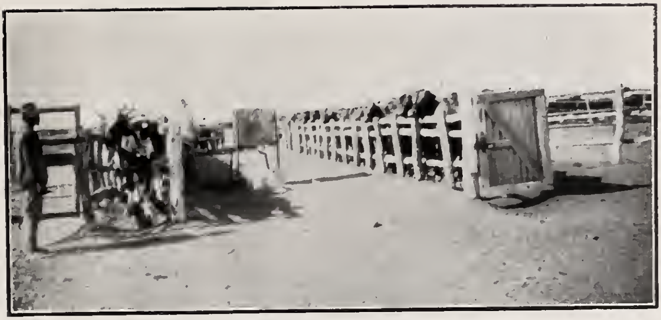


Fig. 25.



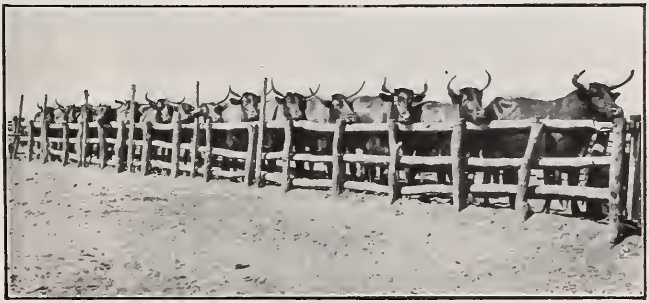
Figs. 24 and 25.—Sussex Bulls.
Sires of the Three-quarter-bred Grade.

Fig. 26.



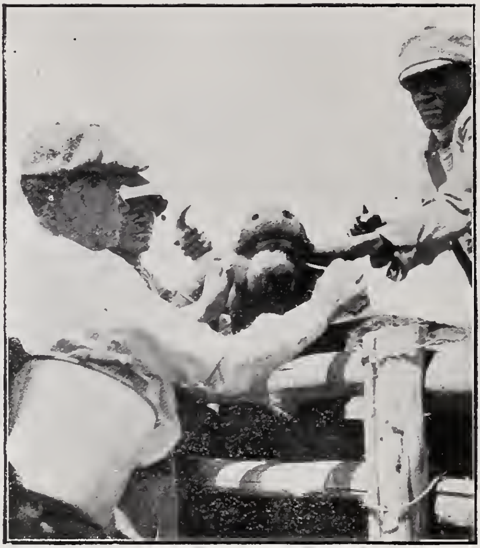
Double Dosing Crushes at Armoedsvlakte.

Fig. 27.



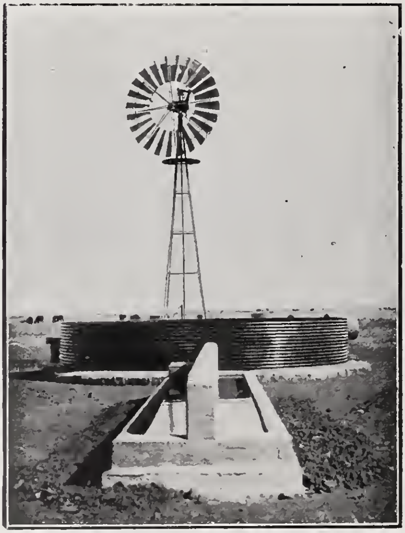
Cattle Ready to be Dosed.

Fig. 28.



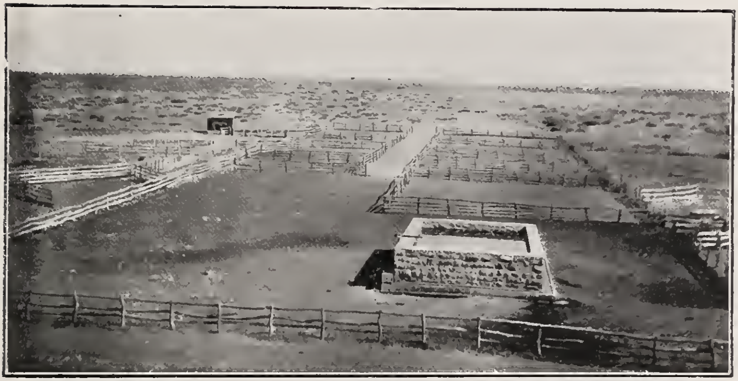
Animal being Dosed with Bone-meal.

Fig. 29.



Water Supply in Camps.

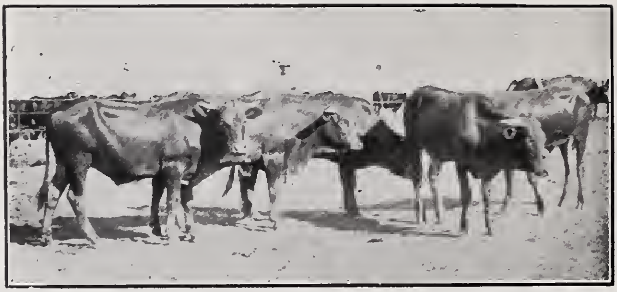
Fig. 30.



Bird's Eye View of Cattle Kraals at Armoedsvlakte.

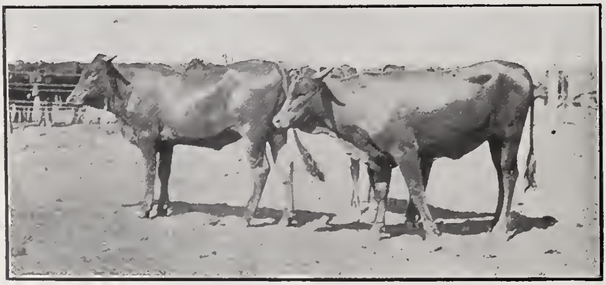
Africander Half-bred Batch I Grade.
(Twelve Months Old.)

Fig. 31.



Bone-meal Tollies.

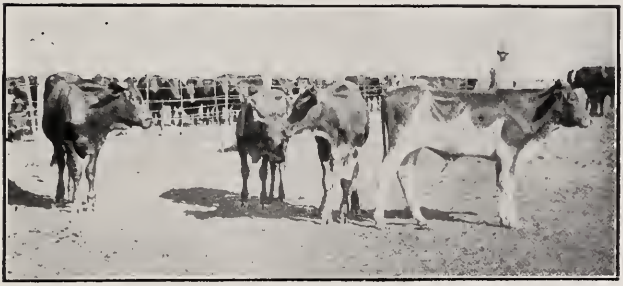
Fig. 32.



Bone-meal Heifers.

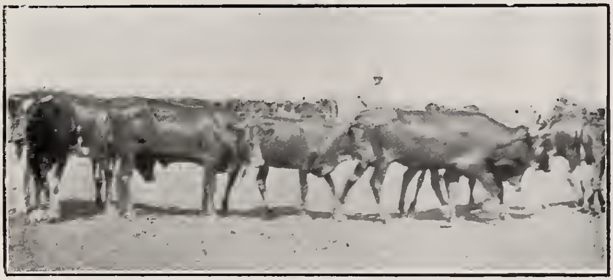
Africander Half-bred Batch 1 Grade.
(Twelve Months Old.)

FIG. 33.



Control Tollies.

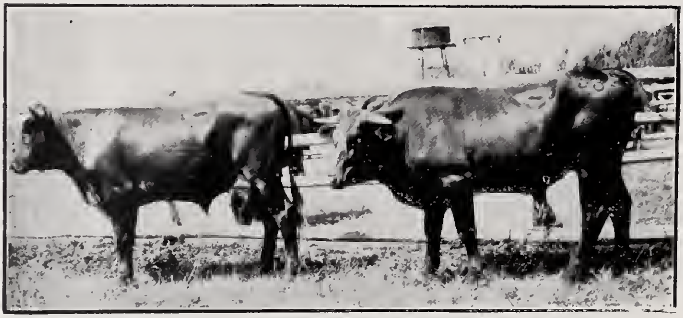
FIG. 34.



Control Heifers.

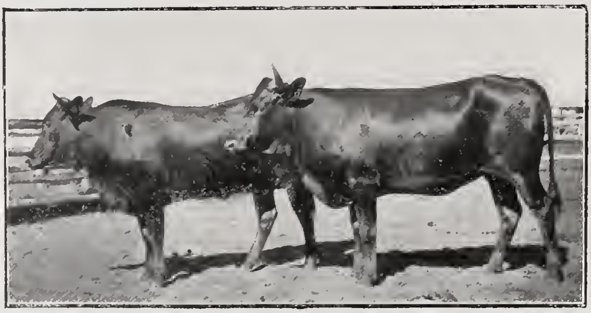
Africander Half-bred Batch I Grade.
(Two Years Two Months Old.)

FIG. 35.



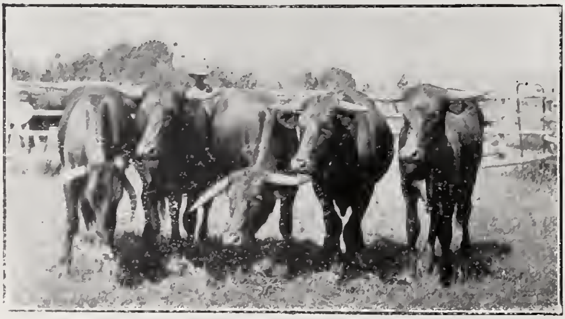
Bone-meal Tollies.

FIG. 36.



Bone-meal Heifers.

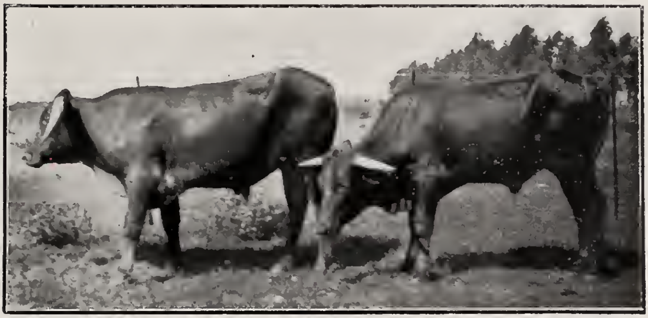
FIG. 37.



Five Bone-meal Tollies.

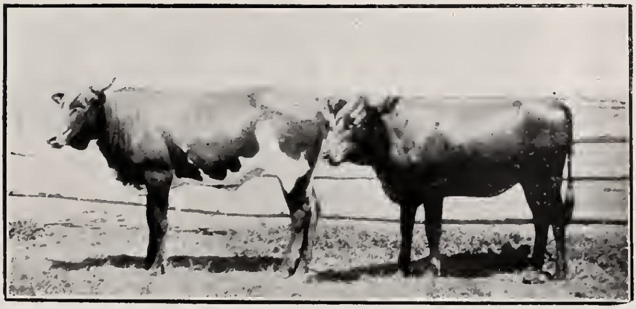
Africander Half-bred Batch I Grade.
(Two Years Two Months Old.)

FIG. 38.



Control Tollies.

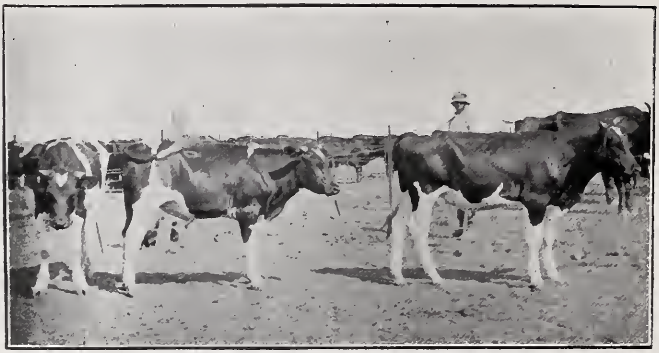
FIG. 39.



Control Heifers.

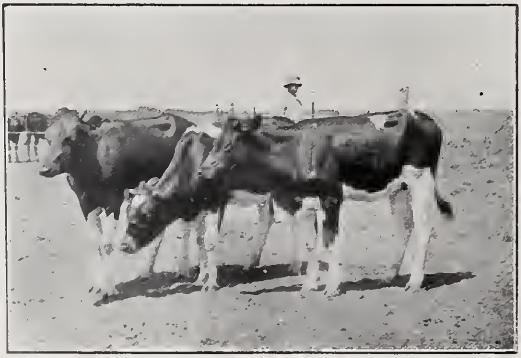
Fries Half-bred Batch I Grade.
(Twelve Months Old.)

FIG. 40.



Bone-meal Tollies. True to Type.

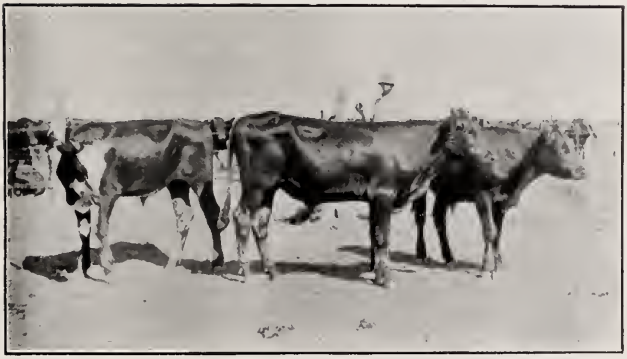
FIG. 41.



Bone-meal Heifers. True to Type.

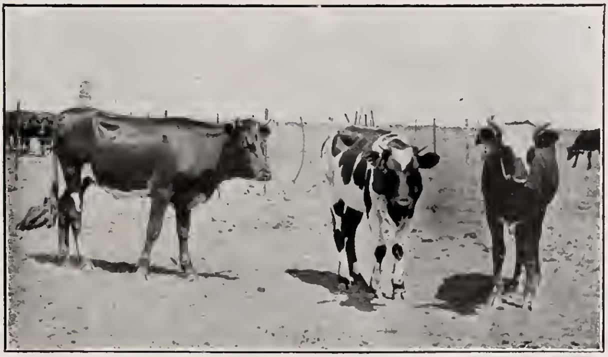
Fries Half-bred Batch I Grade.
(Twelve Months Old.)

FIG. 42.



Bone-meal Tollies. Off Type.

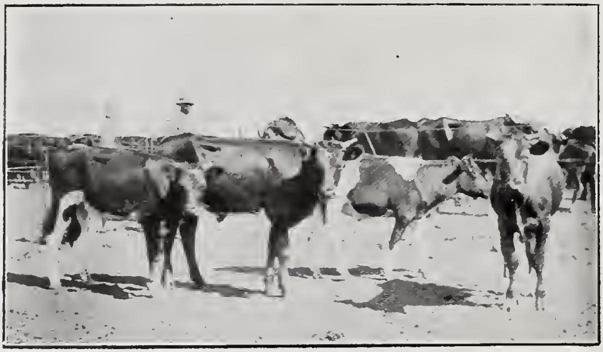
FIG. 43.



Bone-meal Heifers. Off Type.

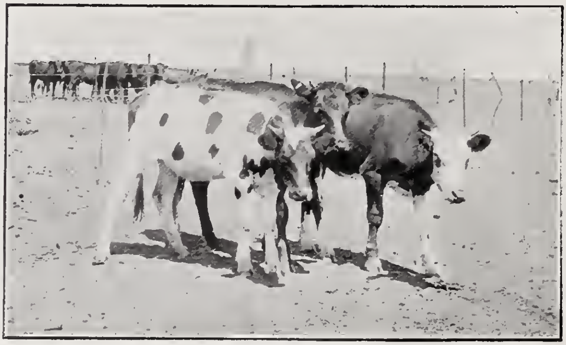
Fries Half-bred Batch I Grade.
(Twelve Months Old.)

FIG. 44.



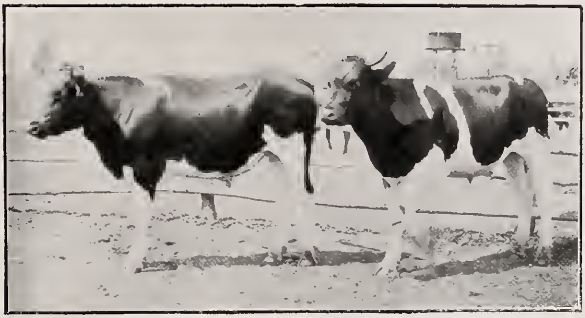
Control Tollies.

FIG. 45.



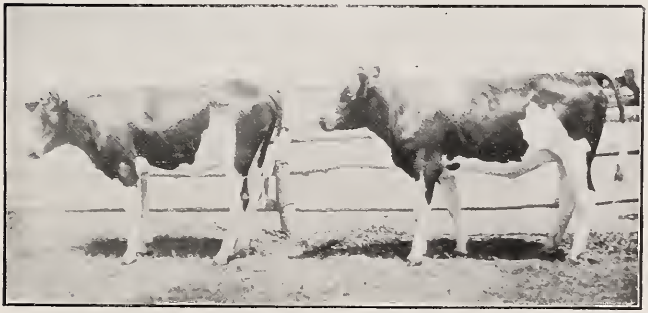
Control Heifers.

Fries Half-bred Batch I Grade.
(Two Years Two Months Old.)
FIG. 46.



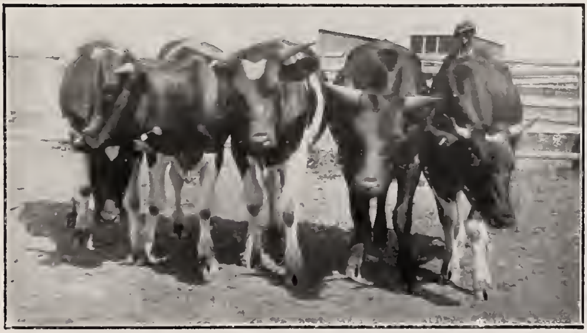
Bone-meal Tollies.

FIG. 47.



Bone-meal Heifers.

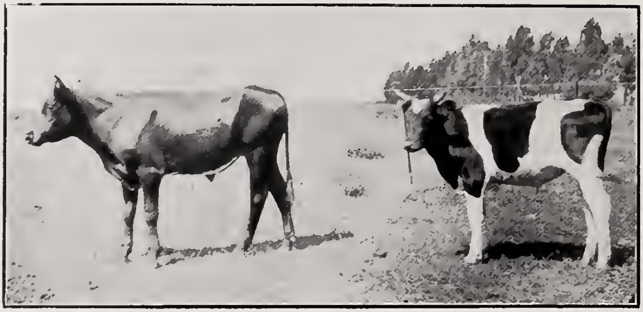
FIG. 48.



Five Bone-meal Tollies.

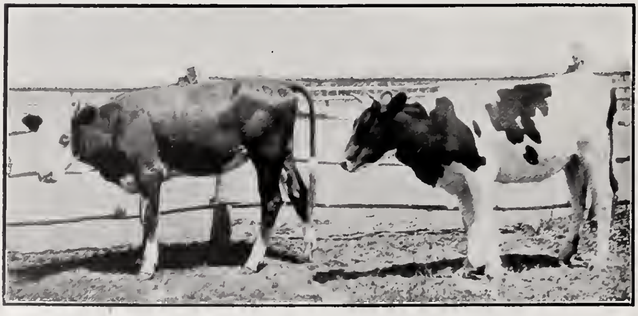
Fries Half-bred Batch I Grade.
(Two Years Two Months Old.)

FIG. 49.



Control Tollies.

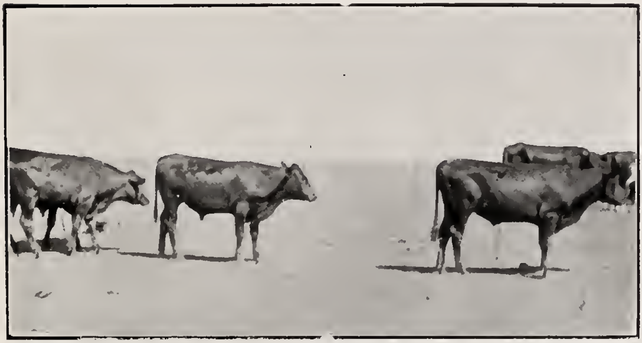
FIG. 50.



Control Heifers.

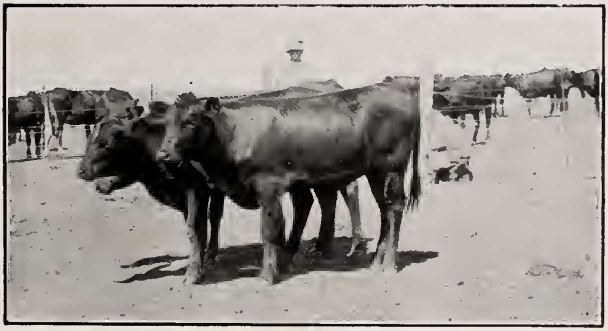
Red Poll Half-bred Batch I Grade.
(Twelve Months Old.)

FIG. 51.



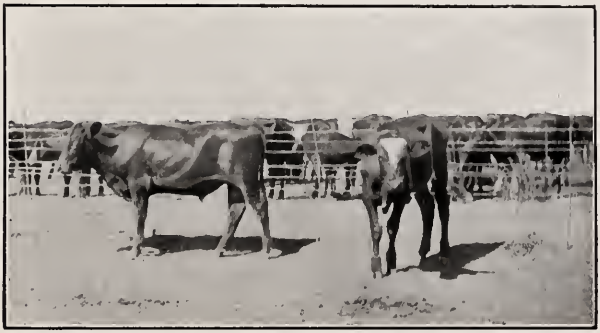
Bone-meal Tollies.

FIG. 52.



Bone-meal Heifers.

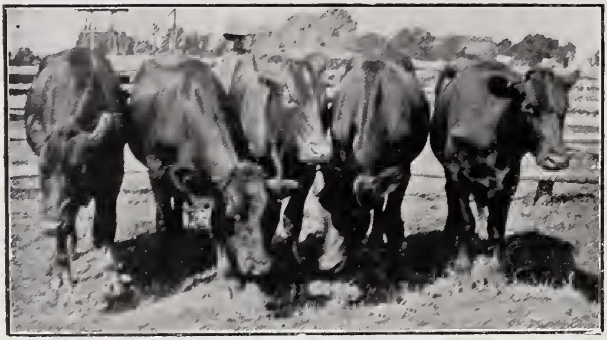
FIG. 53.



Control Tollies.

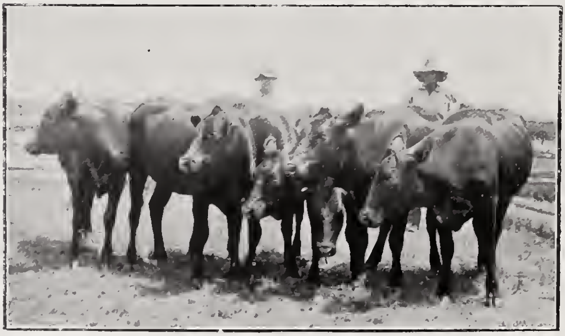
Red Poll Half-bred Batch I Grade.
(Two Years Two Months Old.)

Fig. 54.



Five Bone-meal Tollies.

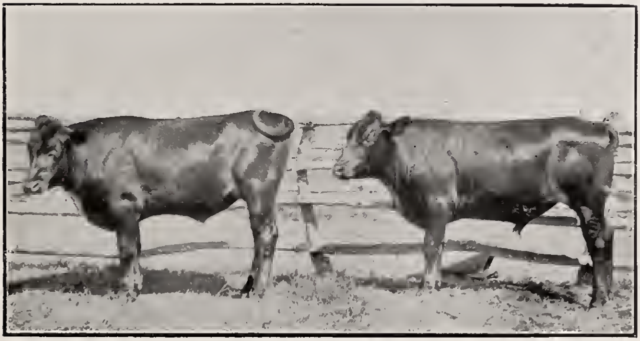
Fig. 55.



Five Bone-meal Heifers.

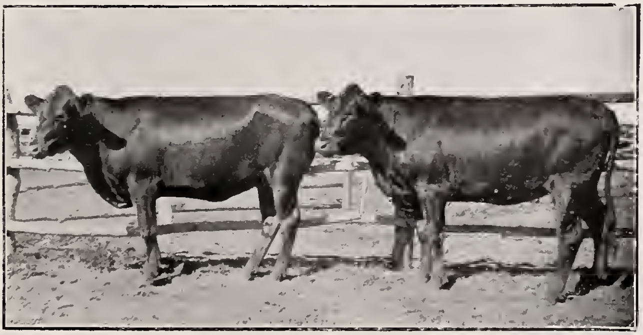
Red Poll Half-bred Batch I Grade.
(Two Years Two Months Old.)

FIG. 56.



Bone-meal Tollies.

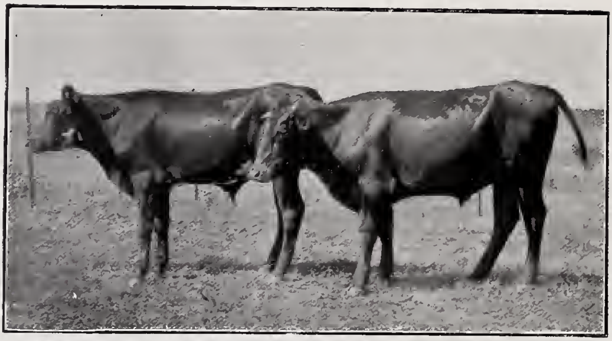
FIG. 57.



Bone-meal Heifers.

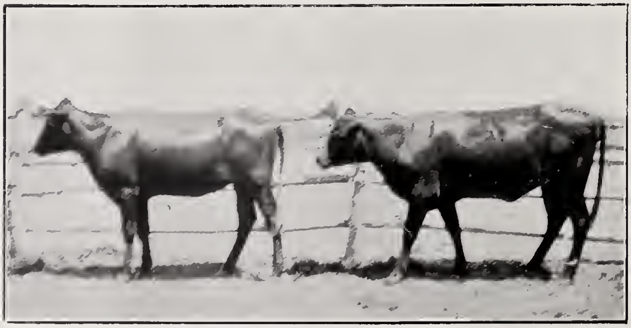
Red Poll Half-bred Batch I Grade.
(Two Years Two Months Old.)

Fig. 58.



Control Tollies.

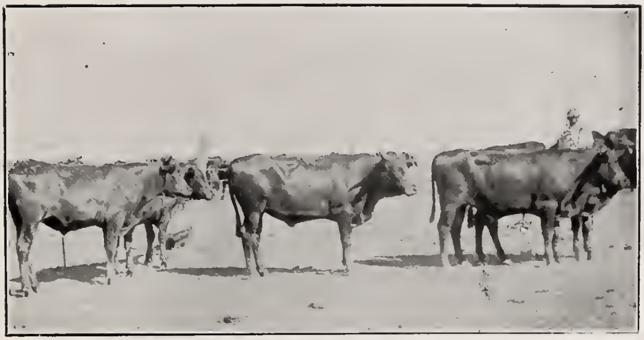
Fig. 59.



Control Heifers.

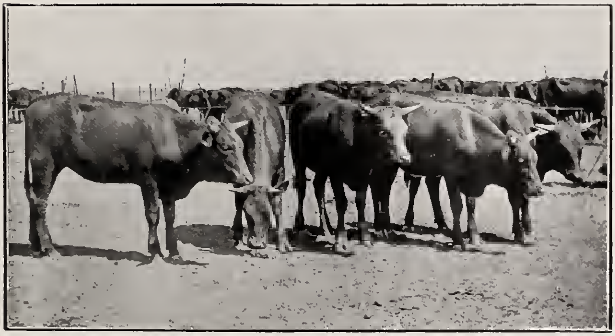
Sussex Half-bred Batch I Grade.
(Twelve Months Old.)

FIG. 60.



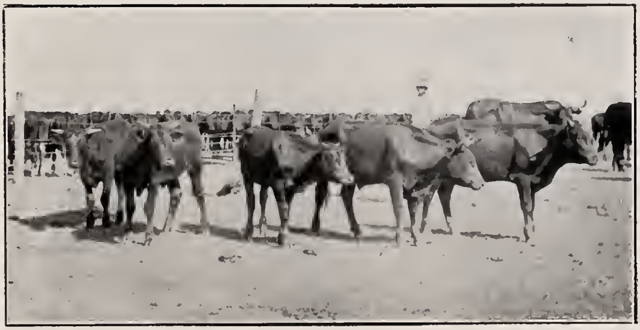
Bone-meal Tollies.

FIG. 61.



Bone-meal Heifers.

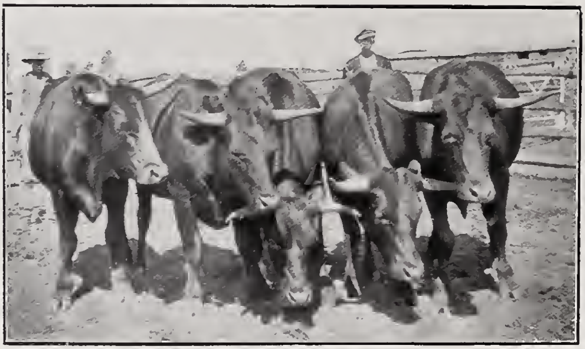
FIG. 62.



Control Tollies.

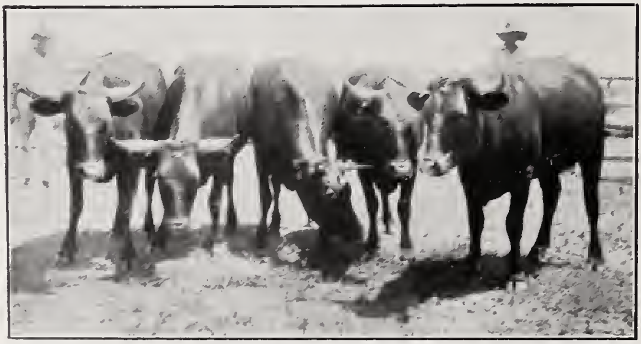
Sussex Half-bred Batch I Grade.
(Two Years Two Months Old.)

Fig. 63.



Five Bone-meal Tollies.

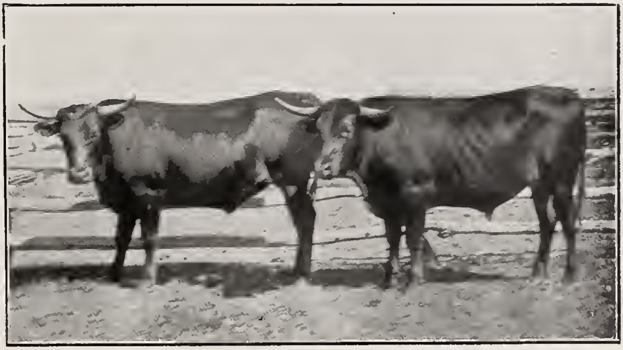
Fig. 64.



Five Bone-meal Heifers.

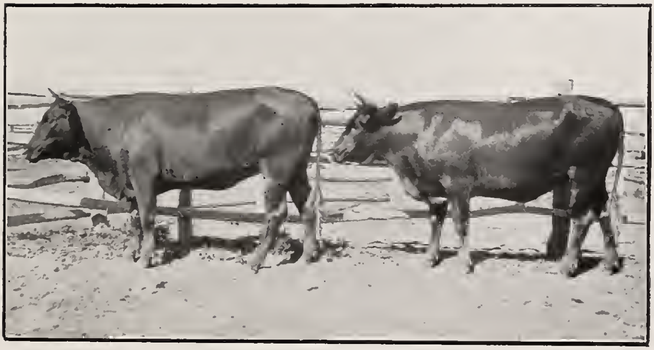
Sussex Half-bred Batch I Grade.
(Two Years Two Months Old.)

FIG. 65.



Bone-meal Tollies.

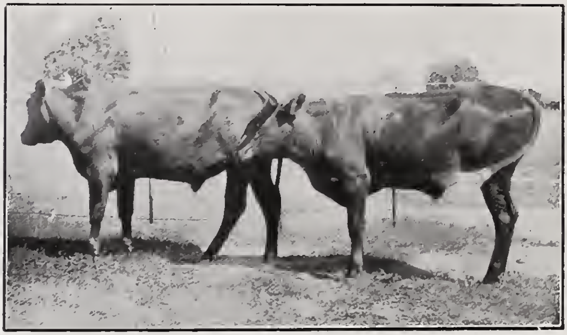
FIG. 66.



Bone-meal Heifers.

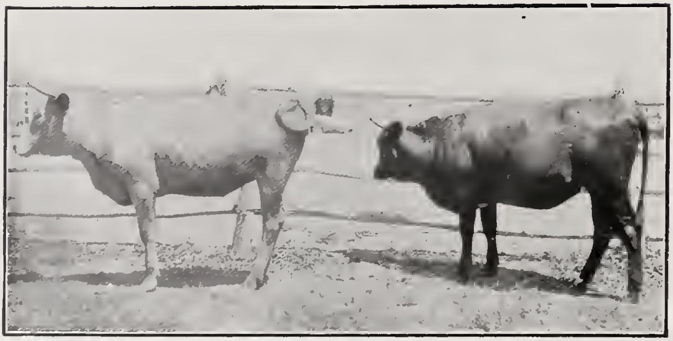
Sussex Half-bred Batch I Grade.
(Two Years Two Months Old.)

FIG. 67.



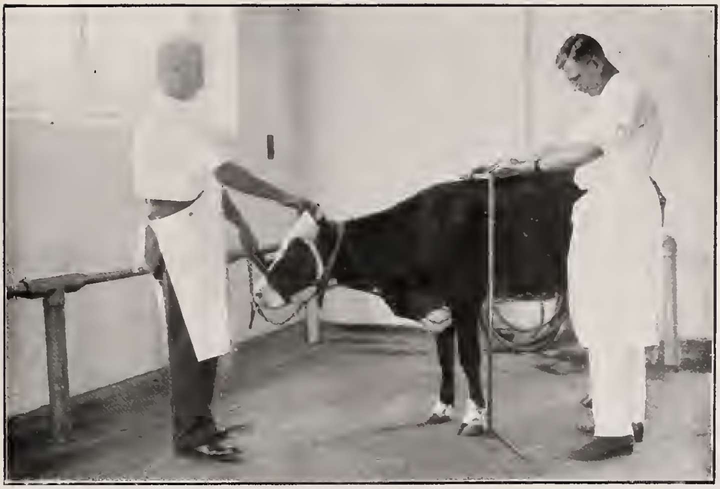
Control Tollies.

FIG. 68.



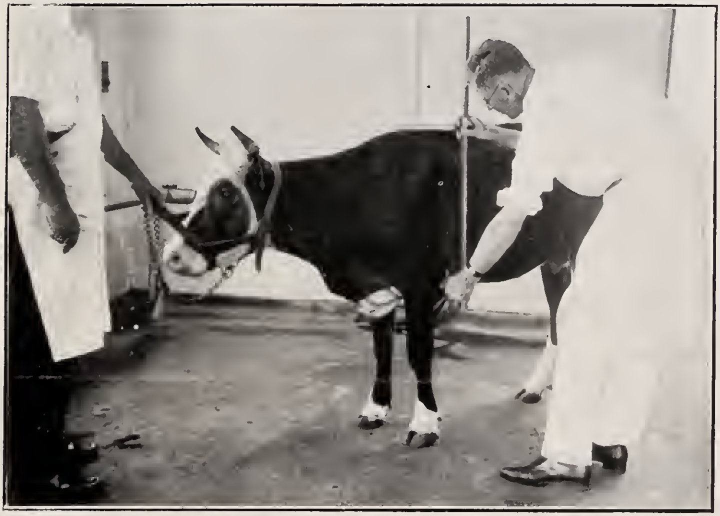
Control Heifers.

FIG. 69.



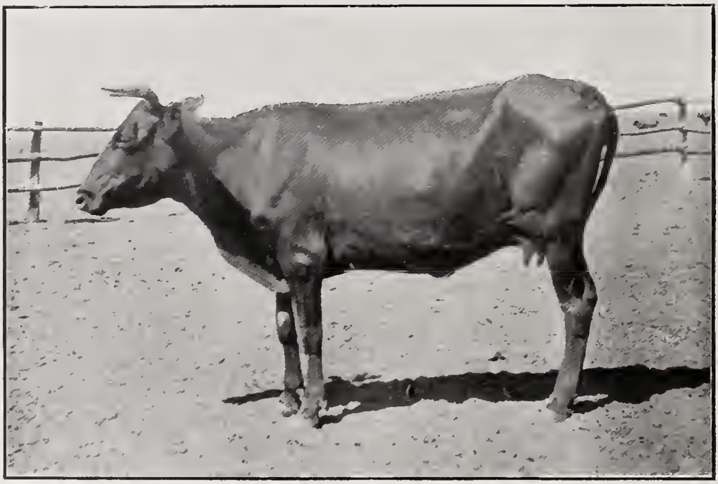
Measuring Cattle with the Deriaz Measuring Stick.
Measuring Height at Withers.

FIG. 70.



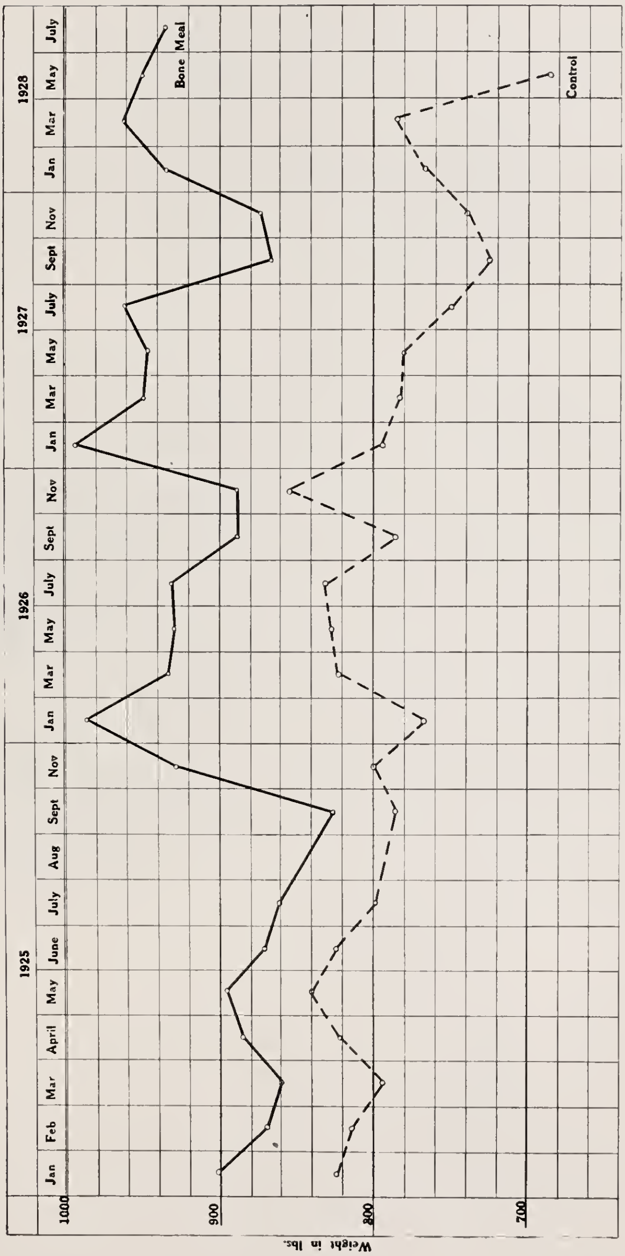
Measuring Depth of Chest.

Fig. 71.



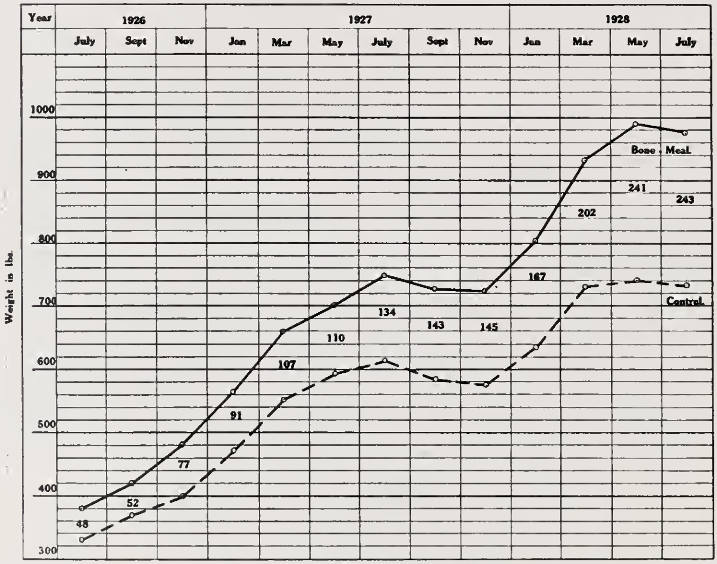
Cow No. 1406. A Control since January, 1925.
Has never been known to show craving for Bone.

GRAPH No 1



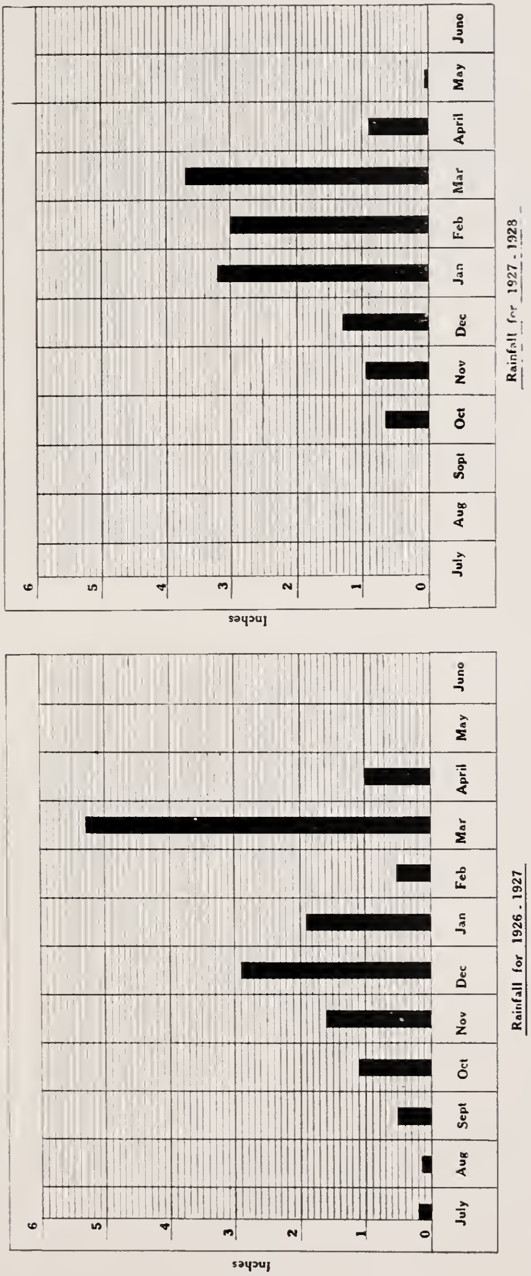
Average weights of Basis Herd Cows

GRAPH No 2.



Average Weights of Half Bred Animals

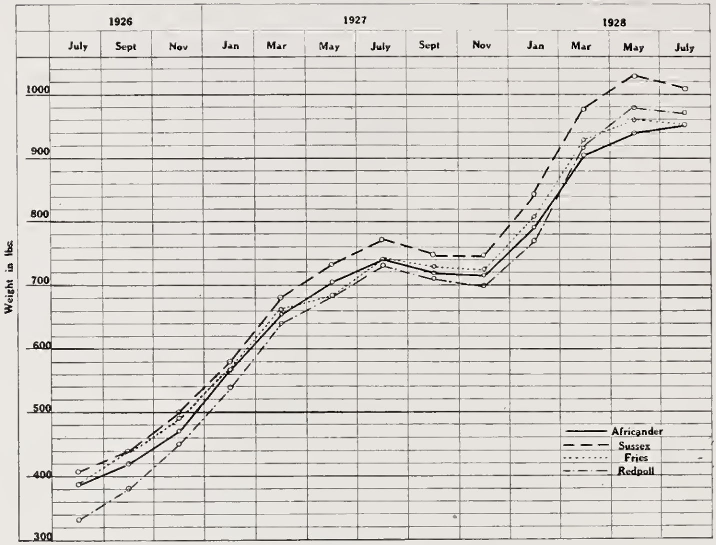
GRAPH No 3



Rainfall for 1927 - 1928

Rainfall for 1926 - 1927

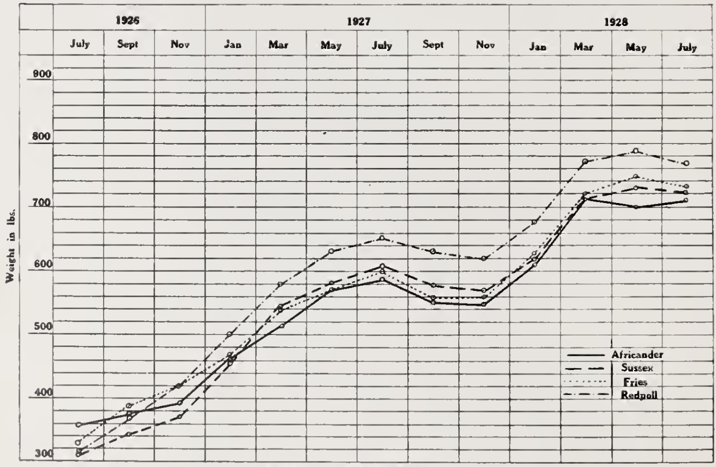
GRAPH No 4



Half-Bred Africander, Redpoll, Fries and Sussex.

BONE MEAL

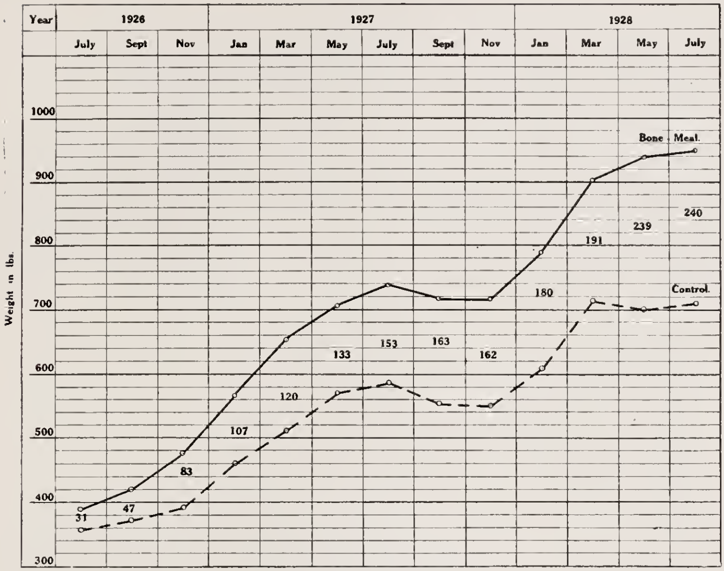
GRAPH No 5



Half-Bred Africander, Redpoll, Fries and Sussex.

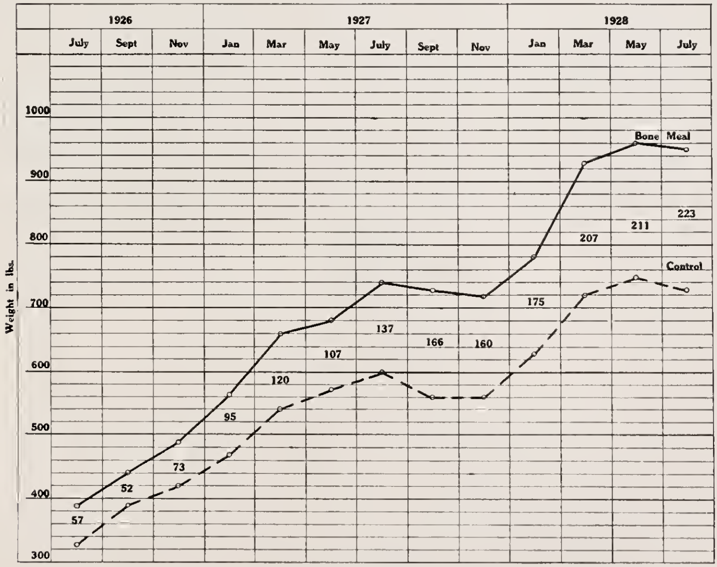
CONTROLS

GRAPH No 6.



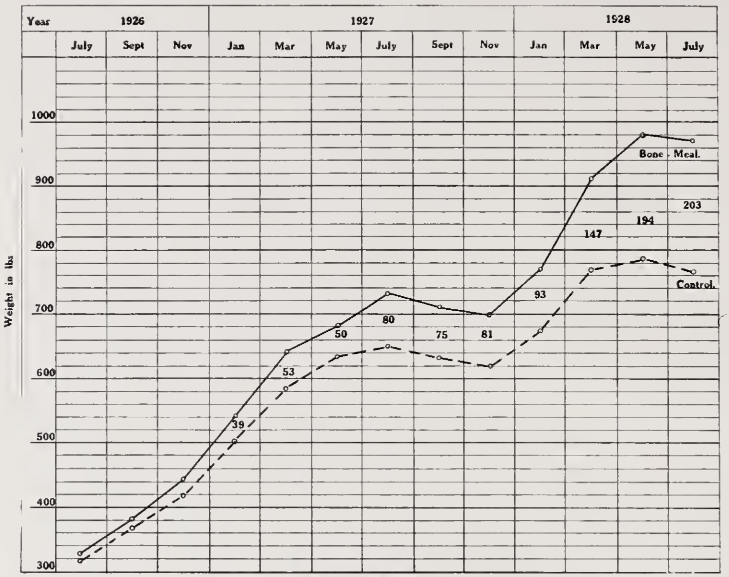
Africander Half-Bred. Batch 1

GRAPH No 7



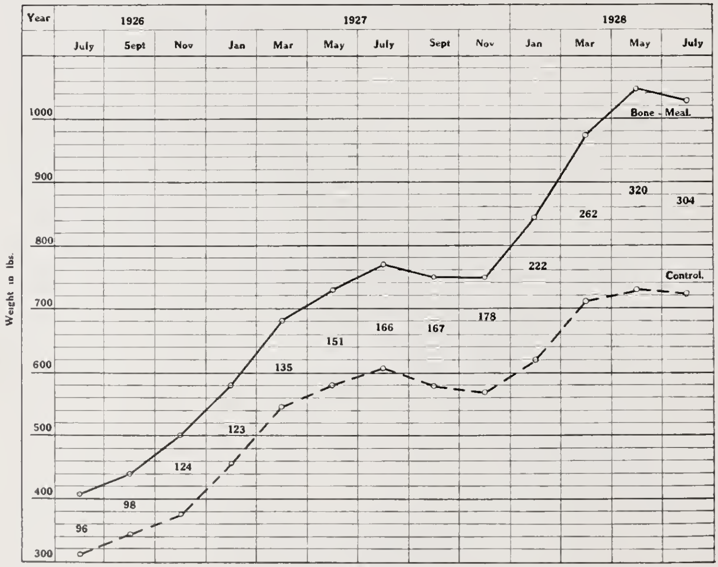
Friss Half-Bred. Batch 1.

GRAPH No 8.



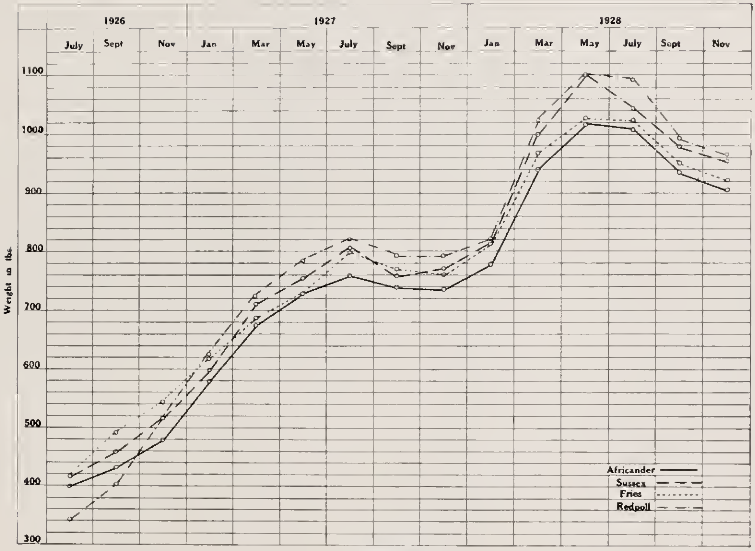
Redpoll Half-Bred. Batch 1.

GRAPH No 9



Sussex Half-Bred. Batch 1

GRAPH No 10

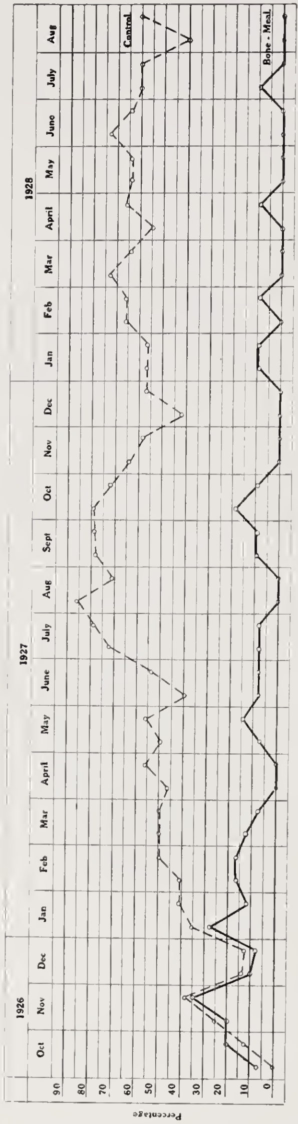


SLAUGHTER TEST

Half Bred Tollies

BONE MEAL

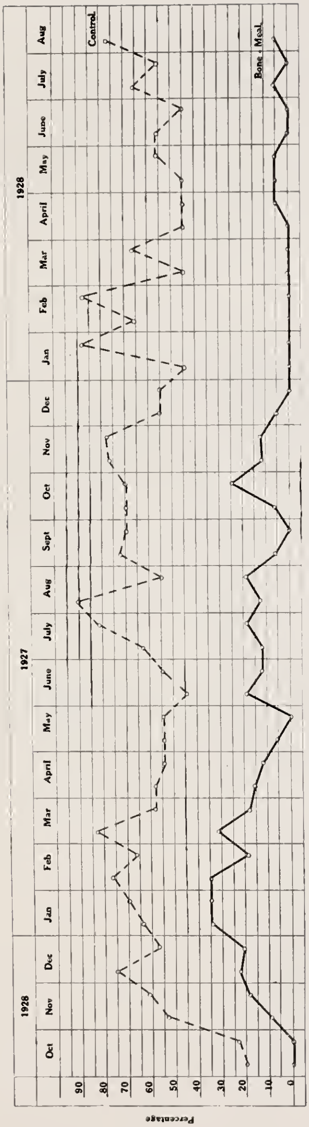
GRAPH No 11



Africander Half-Bred. Batch 1.

Comparative Craving for Rotten Bone.

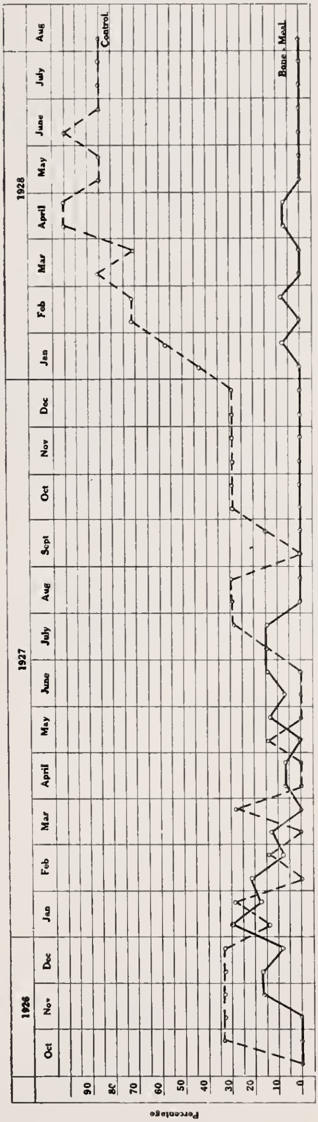
GRAPH No 12



Fries Half-Bred. Batch 1.

Comparative Craving for Rotten Bone.

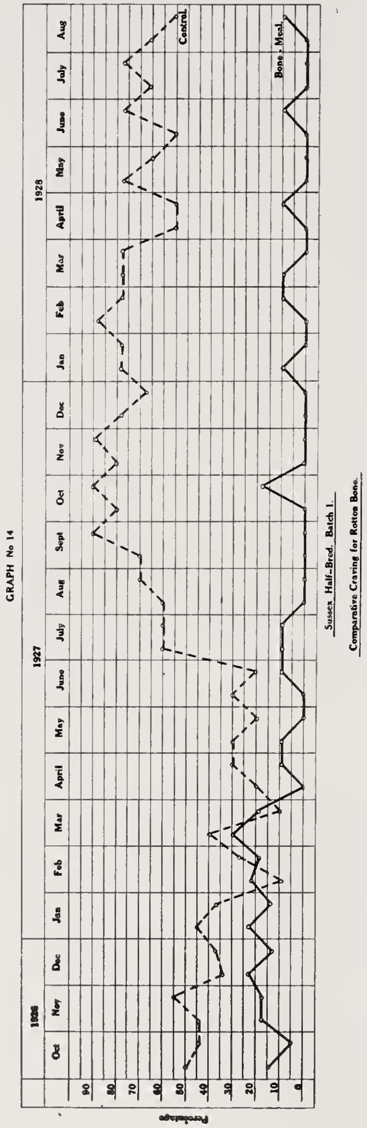
GRAPH No 13



Redpoll Half-Bred. Batch 1.

Comparative Craving for Rotten Bone.

GRAPH No 14



Sussex Half-Bred. Batch 1.

Comparative Craving for Rotten Bone.

Section IX.

Miscellaneous.

G. de Kock ...	Further Observations on the Etiology of Jaagsiekte in Sheep.
H. H. Curson ...	Anatomical Studies No. 7. Hypoplasia of a Testicle and Hyperplasia of the Prostate in a Dog.
J. P. van Zyl ...	On the Toxicity of Arsenic to Fowls.
B. S. Parkin ...	A Clinical Case—Spasm of the Diaphragm in a Horse.
B. S. Parkin ...	The Intra-abomasal Administration of Drugs to Sheep.

15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

Further Observations on the Etiology of Jaagsiekte in Sheep.

By GILLES DE KOCK, M.R.C.V.S., Dr.Med.Vet., D.Sc.,
Sub-Director of Veterinary Services.

UP to the present Jaagsiekte in sheep has been regarded as an infectious disease. Cowdry (1925) studied this disease in South Africa, and in his paper refers to earlier publications. Mitchell (1915) laid aside the parasitic theory, and concluded from his experiments that the conditions of transmission pointed to a specific virus. So far no one has been able to transmit jaagsiekte to healthy sheep by the injection (or inhalation) of blood and diseased tissue.

The following are some of the most important data found in the publications of Mitchell and Cowdry:—

(*a*) Enclosures, in which sheep suffering from jaagsiekte have been kept, constitute sources of infection for incoming sheep;
(*b*) apparently jaagsiekte is transmitted by contact since it appears after the importation of new sheep;
(*c*) the average mortality in infected areas is about 1.6 per cent. per annum, and occurs throughout the year;
(*d*) all ages are affected, but chiefly at 3 years; lambs never;
(*e*) no evidence that the character of the water supply and of the grazing are determinative factors, because jaagsiekte occurs on grass veld, in the Karroo, in the bushveld, and on mixed veld;
(*f*) according to Mitchell, the greatest mortality, however, occurs in particularly wet years, and more cases develop in the flock during the rainy season than during the dry season;
(*g*) cases usually occur singly, and no instance is on record in which large numbers of sheep in the same flock were affected; under natural conditions death occurs in from 2 to 8 months;
(*h*) by netting farms and allowing sheep to graze night and day the mortality has been decreased, i.e. provided all infected animals are slaughtered as soon as symptoms are shown.

Observations recently made and information received from many farmers were in many instances in conflict with the data given by Mitchell and Cowdry. Some of the more important points raised by farmers may be briefly considered.

M. writes from Rosendaal, Orange Free State, that jaagsiekte occurs in 2-4 tooth sheep, and chiefly young rams are affected. In 1928 he lost three valuable rams. They were isolated as soon as symptoms were observed. He has never seen the condition in ewes. M. is of the opinion that jaagsiekte stands in some relation to the regular dosing of Cooper's dip, Theiler's copper sulphate, etc., for parasitism. It is impossible for him to say how infection was introduced on his farm. Rams are bought from time to time at the stud sales. If the disease is infectious, then it must be so only very, very slightly. The rams recently lost by him were related, in fact, two of them were from the same ram.

The Principal, School of Agriculture, Middelburg, writes that as far as jaagsiekte is concerned it was very prevalent three or four years ago, but by adopting a policy of isolating all sheep as soon as they show symptoms of jaagsiekte, it has been reduced to a minimum. The following deaths from jaagsiekte occurred in an average flock of 4,000-5,000 sheep:—

1925-1926	.115	per cent.
1926-1927	.166	,,
1927-1928	.100	,,

M. writes from Graaff-Reinet that in one case where the sire had jaagsiekte one of his lambs died of this disease. He knows of one other case where he sold a ram, and after about six months the ram developed jaagsiekte and died within three weeks after taking ill. This was rather soon, but the fact remains that fully 50 per cent. or possibly more of his progeny died of the same disease when two tooth.

A. M. P., Heilbron, informed the writer in 1927 that jaagsiekte only manifested itself on his farm in 1926. He has been on the farm for 17 years. He is of opinion that the disease was brought on to his farm by stud sheep purchased at the sales. The disease has since only been noted in young stud rams, bred on the farm, and in them the mortality has been relatively high.

V. writes from Griquastad that the disease may make its appearance at any time of the year, and that only full-grown sheep are affected.

The writer visited the School of Agriculture, Tweespruit, Orange Free State, in August, 1927, in connection with the high mortality from jaagsiekte. It was at once felt that this centre would be an ideal place for conducting field observations and carrying out in-contact and other experiments. Facilities were available for the careful control of mortality amongst the sheep on the farm. It was decided to concentrate there more or less on the following points:—

(*a*) Does sex, age, climate, locality, kraaling, diet, breeding time of the year, etc., influence the incidence of jaagsiekte?

(*b*) Is jaagsiekte an ordinary infectious disease?

The greatest difficulty encountered in the study of this disease, and one of utmost importance, was the fact that it was practically impossible to diagnose the disease in its earliest stages, i.e. before clinical symptoms had manifested themselves. It was at once felt that sheep supposed to be healthy and utilized in the various experiments might not be free from disease. Furthermore, it was found impossible to say when a sheep had become infected with jaagsiekte. In order to overcome these difficulties, it was decided to run these experiments for at least 18 months or so, and at the end of each experiment all animals used should be slaughtered in order to ascertain whether any had become infected. By studying the nature of the lesions, it may be possible to indicate whether the lesions were old standing or not. In order to exclude the selection of possible infected sheep for experiments, all animals used were drafted from sheep born at Onderstepoort, and in which no symptoms had been observed.

In the following table a brief summary is given of the incidence of jaagsiekte at Tweespruit:—

CASES OF JAAGSIEKTE AT TWEESPRUIT DURING THE YEAR 1927 AND 1928.

1927 (*about* 800 *Sheep*).

January	1 E	1
February	1 E, 1 E	2
March	1 YH, 1 YE	2
April	1 E. 1 YE, 1 YH	3
May	—	—
June	1 YH, 1 YH	2
July	1 H, 1 2TH. 1 E, 1 H	4
(The majority of the above confirmed microscopically.)		
August	1 E in L, 1 YE, 1 E, 1 YH, 1 H, 1 YH. 1 2TH	7
September	1 E in L, 1 YH, 1 E in L, 1 YE, 1 YH, 1 E, 1 L, 1 YE	8
October	—	—
November	1 2TH, 1 E, 1 H	3
December	1 2TH	1
		33

1928 (*about* 1,000 *sheep*).

January	1 2TH, 1 2TE, 1 YH, 1 YH	4
February	1 2TH, 1 H, 1 L	3
March	—	—
April	1 L, 1 4TH, 1 2TH, 1 2TH, 1 YH, 1 2TH, 1 YH	7
May	1 2TH, 1 4TH	2
June	—	—
July	1 E, 1 YH, 1 H	3
August	1 H	1
September	1 YE, 1 4TH	2
October	1 YH	1
November	1 H	1
December	—	—
		24

(All cases since August, 1927, confirmed microscopically.)

Abbreviations.

L = Lamb.
E = Ewe.
H = Hamel.
Y = Yearling.
2T, 4T = Two-tooth, four-tooth, etc.

In the following table an attempt has been made to indicate how the different sexes, ages, etc., were affected with jaagsiekte at Tweespruit in 1927 and 1928:—

	Lambs.	*Hamels.*	*Ewes.*
Lambs	3	—	—
Yearling Hamels	—	14	—
2-Tooth Hamels	—	9	—
4-Tooth, and over, Hamels.	—	10	—
Yearling Ewes	—	—	6
2-Tooth Ewes	—	—	2
4-Tooth, and over, Ewes..	—	—	10
Ewes in Lamb	—	—	3
	3	33	21

From this table it will be seen that 25 two-tooth and under were affected, whereas 32 were over two-tooth. It would appear that young sheep are as liable to be affected as old ones. The disease was definitely recognized in a lamb 2½ months old. In 1927 the mortality at Tweespruit was in the vicinity of 4 per cent., whereas in 1928 it was below 2½ per cent. In 1927 the different sexes in the Tweespruit flocks were more or less distributed as follows:—

260 Ewes;
135 Hamels;
425 Ewes in lamb.

When the percentage mortality in the different sexes is considered for 1927, it will be seen that about 25 per cent. of the hamels were affected, whereas in ewes it was only about 3 per cent. From the information received from farmers it would appear that rams are even more susceptible than hamels.

As pointed out above, it is impossible to say definitely when an animal had become affected with jaagsiekte. Clinical manifestations of the disease may only appear some time after lesions had formed in the lungs. It would be highly desiralbe to refer here to one important observation made by the writer during his investigations. In the case of the sheep used for the in-contact experiments at Tweespruit and at Onderstepoort, no clinical symptoms were noticed in any of them while they were under observation for a period of eighteen months. They were in good condition when they were slaughtered at the Pretoria Abattoirs at the end of the experiment. Amongst these sheep three were found with small localized jaagsiekte lesions. In each case only a small focus was found in one lung. In one case they were so insignificant that they could easily have been overlooked.

In view of these considerations, it is, therefore, almost impossible to say at what time of the year the disease was most prevalent at Tweespruit. However, if the climatic conditions at Tweespruit are considered, it would appear that jaagsiekte made its appearance in an area relatively dry during the summer and fairly cold during the winter. One can definitely say that the disease in this locality was not influenced by wet weather.

From the records of the specimens and the sheep received at Onderstepoort for diagnosis, cases have occurred in the following localities:—

Cape Province and Bechuanaland.

(Karroo Veld): Belmont, Carnarvon, De Aar, Aliwal North, Graaff-Reinet, Middelburg, Beaufort West.
(Mixed Veld): Taungs, Vryburg, Douglas, Petrusburg.
(Coastal Belt): Umtata.

Orange Free State.

(Grass Veld): Bloemfontein, Kaffersdam, Bestersput, Ladybrand, Steynsrust, Postmasburg, Rosendaal, Heilbron, Vredefort.

Transvaal.

(Grass Highveld): Amersfoort, Devon, Nooitgedacht (Ermelo), Kinross.
(Lowveld Mixed Grazing): Lichtenburg, Waterberg.

It will be seen that the disease is liable to make its appearance on all types of veld in the Transvaal, Orange Free State, and Cape Province. No cases are recorded from Natal. It is, therefore, impossible to say whether the disease occurs in that Province. It is quite possible that jaagsiekte cannot be distinguished by farmers from sporadic pneumonia in sheep. Furthermore, in view of the low percentage mortality, it is therefore most likely that the disease may have been overlooked in Natal. It would appear that more cases are recorded on Karroo veld (Cape) and grassveld (Orange Free State), and highveld (Transvaal). It is possible that the disease has been recognized in newly introduced sheep in other centres, but that it was not able to establish itself there. The high incidence of the disease in certain areas may be due to the fact that these districts have for years been the recognized sheep centres of the Union, and where for years sheep breeding has been carried on. In the other centres the disease has not had an opportunity to establish itself. The writer is, however, of opinion that environment may play a part in the incidence of the disease, perhaps as an exciting cause.

In going through the Onderstepoort records it was found that a large number of cases occurred amongst Karroo sheep introduced for the preparation of blue-tongue vaccine. In one instance a sheep died of the disease 46 months after its arrival at Onderstepoort. It should be pointed out that these Karroo sheep should have left their destination in a clinically healthy state, but affected cases have undoubtedly been overlooked in view of the death of some of the animals soon after arrival. The following table will indicate the incidence of jaagsiekte in these introduced sheep:—

District from which Purchased.	Number of Sheep Purchased.	Eartag Number of Sheep affected.	Date of Arrival.	Period at Onderstepoort before Death.
Beaufort West.........	—	7540	20/7/23	16 months.
		7405	20/7/23	46 months.
		16344	22/3/27	4 months.
Middelburg, Cape.......	500	9598	14/7/24	5 months.
		9374	14/7/24	6 months.
		9155	9/7/24	13 months.

District from which Purchased.	Number of Sheep Purchased.	Eartag Number of Sheep affected.	Date of Arrival.	Period at Onderstepoort before Death.
Philipstown...........	1,000	10641	19/12/24	27 months.
	(1924)	10582	19/12/24	33 days.
		10691	20/12/24	29 days.
		10843	20/12/24	25 days.
	1,500	11092	14/ 1/25	1 day.
	(1925)	11035	14/ 1/25	12 days.
		11090	14/ 1/25	20 days.
		10986	14/ 1/25	21 days.
		11117	14/ 1/25	9 months.
		11666	20/ 3/25	1½ months.
		12975	31/ 8/25	9 months.
	1,000	14312	14/ 1/26	2 months.
	(1926)	15005	8/ 9/26	3 months.
		15754	17/12/26	1 day.
	600	17149	9/ 6/27	6 days.
	(1927)	17340	15/ 6/27	2 months.

There can be no doubt that infected animals were introduced, seeing that sheep succumbed soon after arrival, and that this interval varied from one day to several days, to several weeks, to several months, and even to 27 and 46 months respectively in two cases. Is it possible for an animal to remain alive, or harbour jaagsiekte lesions for considerable periods before symptoms appear, or did some of the animals contract the disease after their arrival at Onderstepoort? In this respect the following cases may be cited where animals were definitely introduced as infected animals for experimental purposes, and used for considerable periods before they died of jaagsiekte or were destroyed *in extremis*:—

Eartag No. of the Sheep.	No. of the Specimen.	Date of Arrival.	Date of Death.	Interval between Date of Arrival and Date of Death.
17939	7329	2/ 8/27	Died 9/ 9/27	38 days.
18339	7489	26/ 8/27	Killed 5/11/27	About 3 months.
18340	7781	26/ 8/27	Died 26/ 2/28	6 months.
18371	7503	20/ 9/27	Died 12/11/27	About 2 months.
18592	7817	11/11/27	Killed 7/ 3/28	4 months.
18531	—	26/10/27	Killed 27/ 3/29	17 months.
19646	8241	2/ 5/28	Died 23/ 7/28	2 months.
19642	8387	30/ 4/28	Died 6/ 9/28	5 months.
19643	8485	30/ 4/28	Died 27/ 9/28	5 months.
21635	—	23/ 8/28	Still alive 23/ 1/29	5 months.
21636	—	23/ 8/28	Still alive 23/ 1/29	5 months.

N.B.—Sheep 18531, Ram: Arrived at Onderstepoort on the 26.10.27 from Heilbron, O.F.S., and used for a jaagsiekte in-contact experiment. The animal was in poor condition and when driven showed marked respiratory distress.

12.4.1928: Condition poor; marked respiratory distress still present when the animal was driven. Definite changes in the left lung could be diagnosed.

4.6.1928: Condition poor, but the animal showed slight improvement in condition.

27.3.1929: The animal showed marked improvement in condition, no respiratory distress when the animal was driven and no signs of ill-health seen. The animal was destroyed for post-mortem in order to ascertain whether jaagsiekte lesions had been present or were present, seeing that the animal was forwarded to Onderstepoort suffering from jaagsiekte.

Post-mortem revealed about 20 fine shotlike greyish white transparent nodules about 1 mm. in diameter in both lungs. In the left lung there was a large firm nodular swelling about the size of a hen's egg. This was of the nature of an advanced fibrosis. This apparently was a jaagsiekte focus which had partly healed out.

Histologically (Specimen No. 9093), well-formed papilliform cystadenomata could be identified in the fibrous nodule. The multiple shotlike nodules were of the nature of jaagsiekte foci, and in places the proliferations were very prominent. In view of the connective tissue present in these nodules they cannot be regarded as young jaagsiekte lesions.

This therefore is definitely a case of jaagsiekte which had shown definite symptoms before it was admitted here on the 26.10.27. At Onderstepoort it was kept under observation for about 17 months. It would therefore seem that sheep may be carriers of jaagsiekte lesions for considerable periods before they die of the disease or are killed *in extremis*.

The above table clearly indicates, therefore, that sheep may remain alive for considerable periods after they have actually shown symptoms. Ram 18531 is still in good condition, and symptoms of increased and laboured respiration can be identified when the animal is driven. It is being used for a breeding experiment and will be killed in the course of 1929 to substantiate the diagnosis of jaagsiekte. It may be pointed out that in all the above cases where the animals died, or were killed *in extremis*, jaagsiekte was diagnosed microscopically.

Is it possible from the post-mortem report and the microscopical examination of the lesions in the lung to say how long an animal could have been infected?

It would be extremely doubtful to estimate the time limit in those cases exceeding a number of months. Up to that time a comparison can be made with those cases which have succumbed after five months. It must, however, be remembered that some of these cases which died at Onderstepoort may already have been infected for some considerable time on the farm from which they came. Moreover it was shown above that three sheep had definite lesions of jaagsiekte, but so small and localized that they did not reveal any symptoms. Taking all these points into consideration, it seems to be possible that some cases may be affected with jaagsiekte lesions for a considerable period (probably years) before the disease brings about the death of the animal or necessitates its slaughter. Furthermore in view of the neoplasm theory suggested by De Kock (1929), such an extended course may therefore be expected.

With reference to the question of sheep contracting the disease in kraals, it may be stated that on this point a good deal of information was obtained at Tweespruit. It was pointed out above that the incidence of jaagsiekte at Tweespruit is probably the highest yet recorded in South Africa. Yet sheep are allowed to graze day and night and only in some instances are sheep placed in a wire-fenced kraal situated near the homestead. This kraal is more of the nature of a small open camp which allows a large amount of space between the sheep brought there at night. Sheep, not grazing, lie together in groups, even if they are allowed to graze night and day in jackal-proof camps. The conditions present in this Tweespruit camp are therefore more or less similar to those found in the large camps. If jaagsiekte is therefore an infectious disease by mere contact, then it must be contracted far more readily and to a much larger extent where susceptible sheep come into contact with infected ones under the above conditions.

The argument is brought forward that the incidence of jaagsiekte becomes markedly reduced when sheep are allowed to graze day and night in jackal-proof camps. At the same time, however, the practice is introduced of killing all sheep showing symptoms of jaagsiekte. The probable explanation of this decreased mortality is that breeding with infected animals is being reduced to a minimum. This will be more fully discussed later on.

Is Jaagsiekte an Ordinary Infectious Disease?

From the above it will be realized that very little definite information is available regarding the causation of jaagsiekte in sheep. Mitchell maintained that he produced " true " jaagsiekte by contact, but unfortunately his evidence cannot be accepted at present, in view of the information obtained after his investigations. He pointed out that the susceptible sheep used for his experiments were drafted from an area where jaagsiekte had never been found, and, previous to the experiments with jaagsiekte, had been used at the Laboratory for the production of blue-tongue vaccine. From the records it was ascertained that the sheep used by Mitchell in 1912 came from Middelburg, Cape, and in the lung specimens subsequently received from that area jaagsiekte was definitely diagnosed. Furthermore, it was shown above that clinically healthy animals affected with jaagsiekte lesions may be introduced from infected areas. Moreover in view of the short incubation period encountered by Mitchell in his experimental cases, it is therefore more than likely that Mitchell did not transmit jaagsiekte by contact, but that he was dealing with some other infectious pneumonia of sheep which in some of his cases became superimposed on true jaagsiekte lesions.

In view of these discrepancies, several experiments were planned to study the etiology of jaagsiekte. A number of these experiments are still in progress, and will be more fully reported later on. The following are some of the points studied:—

(1) can jaagsiekte be transmitted by direct contact?

(2) if not, are there any predisposing causes?
(3) what are the exciting causes? Can one or other of the following be incriminated, viz.: organisms, mechanical injuries (e.g. dust), etc.?

The following experiments have been undertaken to ascertain whether jaagsiekte can be set up by contact:—

(*a*) Experiment S. 3156 was commenced on the 3rd August, 1927, in order to ascertain whether jaagsiekte could be transmitted by placing susceptible sheep in very close contact (in a small stye) with known infected animals. Infected animals in these experiments came mainly from Tweespruit, and were from time to time introduced into this experiment to replace those that had died or were killed *in extremis*. The histo-pathology of all cases that died was carefully considered. The susceptible animals were always in close contact with at least two infected animals. The following infected sheep were used:—

S. 17938
S. 17939
S. 18339
S. 18340
S. 18371
S. 18373
S. 19607
S. 19608
S. 19639
S. 19640
S. 19643

The following are the numbers of the susceptible sheep employed along with details giving the dates of disposal:—

16530	Dicharged 26.10.27.
16691	
16536	
16537	Killed 22.9.27.
16697	Discharged 26.10.27.
14058	Killed at the Abattoirs on 3.1.29.
14233	
14524	
15344	
15678	
15967	
16702	Killed 18.3.28.
20231	(In contact from 2.7.28.) Killed 9.11.28.
21298	Lamb born July, 1928.
21649	Lamb born September, 1928.

These sheep were in contact for periods varying from a few months up to 17 months. Only one (14524) of these sheep showed slight localized jaagsiekte lesions, and, further, no clinical manifestations were observed.

(*b*) Experiment S. 3188 was commenced on the 26.8.27 in order to ascertain whether jaagsiekte could be transmitted by placing susceptible sheep in contact in an open camp (about 20 yds. × 15 yds.) with known infected animals.

The following infected sheep were utilized:—

18339
18590
19642
21636
18340
18591
19641
18371
18592
19646
18372
19482
19647
18531
19643
21635

The majority of the following susceptible sheep were in contact with infection for periods up to 17 months:—

14226
15977 (In contact from 1.5.28.) Killed at Pretoria Abattoir 3.1.29.
16699
12164 Killed 27.10.27.
14506
14513
14565
14568
15971
16011
16028
16532
16684 Killed at Pretoria Abattoirs 3.1.29.
16703
18001
18341
18348
18349
18528
18529
20231

Lamb—
21291
21292 In contact from July, 1928.
21297
21396 In contact from August, 1928. Killed 8.11.28.

None of the sheep revealed any lesions in their lungs whatsoever when destroyed.

(*c*) Experiment S. 3208 was commenced on the 18th September, 1927, at Tweespruit, where jaagsiekte mortality was approximately 2-4 per cent. This experiment was undertaken to ascertain:

(i) whether specially selected sheep from Onderstepoort would contract jaagsiekte at Tweespruit when exposed to ordinary veld conditions;
(ii) whether such Onderstepoort animals would contract jaagsiekte if allowed to run with those batches of sheep at Tweespruit in which heavy mortality was occurring.

On the 18th September, 1927:

20 Onderstepoort sheep were drafted into (i) and
40 Onderstepoort sheep were drafted into (ii).

On the 12th March, 1928:

50 Onderstepoort sheep were drafted into (i),
60 Onderstepoort sheep were drafted into (ii).

Full particulars about these Onderstepoort sheep will be found in the following tables. All the remaining Onderstepoort sheep at Tweespruit were returned to Onderstepoort on the 7.12.28. Some died at Onderstepoort shortly after their arrival, whereas the majority were slaughtered at the Pretoria Abattoirs.

ONDERSTEPOORT SHEEP SENT TO TWEESPRUIT ON THE 18.9.27 AND RETURNED ON 7.12.28.

Batch 2 (In-contacts).						Batch 1 (Controls).		
12163	X	29/ 5/28	12037	X	21/10/27	9877	A	3/ 1/29
14077	Stolen		12154	A	3/ 1/29	9967	A	20/12/28
14229	X	2/ 7/28	12167	Stolen		11895	X	19/ 9/27
14503	A	3/ 1/29	14046	X	30/ 5/28	11930	X	20/ 9/27
14534	A	20/12/28	14522	A	20/12/28	14081	A	20/12/28
14566	A	3/ 1/29	14529	A	20/12/28	14086	A	3/ 1/29
15674	A	20/12/28	14532	A	20/12/28	14091	A	20/12/28
15752	A	3/ 1/29	14576	A	20/ 1/29	14221	A	20/12/28
15898	A	20/12/28	15699	A	3/ 1/29	14222	X	22/12/27
15969	A	20/12/28	15974	A	3/ 1/29	14225	X	24/10/27
15972	A	20/12/28	15975	A	20/12/28	14230	X	20/12/27
15985	X	?	15997	A	20/12/28	14231	A	3/ 1/29
15998	A	3/ 1/29	16007	A	20/12/28	14520	A	20/12/28
16002	A	20/12/28	16017	A	20/12/28	14525	A	20/12/28
16004	A	20/12/28	16069	A	3/ 1/29	14574	A	20/12/28
16014	A	20/12/28	16101	A	3/ 1/29	15397	A	3/ 1/29
16129	A	20/12/28	16130	A	3/ 1/29	15676	A	20/12/28
18366	A	3/ 1/29	18367	A	3/ 1/29	15962	A	3/ 1/29
18368	Stolen		18370	Stolen		16003	A	20/12/28
14535	A	20/12/28				16022	A	3/ 1/29
						18369	A	3/ 1/29

Abbreviations used :
X = Died.
A = Killed at the Abattoir, Pretoria ; date of death also given.

ONDERSTEPOORT SHEEP SENT TO TWEESPRUIT ON THE 12.3.28 AND RETURNED ON 7.12.28.

Batch 1 (Controls).						Batch 2 (In-contacts).		
19322	A	20/12/28	19360	A	3/ 1/29	19325	A	3/ 1/29
19326	X	10/12/28	19361	A	20/12/28	19327	A	3/ 1/29
19331	A	3/ 1/29	19362	A	3/ 1/29	19328	Stolen	
19335	A	20/12/28	19363	A	20/12/28	19329	Stolen	
19336	A	20/12/28	19367	A	20/12/28	19333	X	29/11/28
19337	A	20/12/28	19369	A	20/12/28	19339	X	10/ 9/28
19338	A	3/ 1/29	19371	X	10/12/28	19342	X	20/ 6/28
19340	X	14/ 8/28	19372	X	15/12/28	19349	A	3/ 1/29
19346	A	3/ 1/29	19376	X	11/12/28	19350	A	3/ 1/29
19347	X	9/ 8/28	19377	A	3/ 1/29	19351	X	3/ 7/28
19348	A	20/12/28	19379	X	9/12/28	19364	A	3/ 1/29
19353	A	3/ 1/29	19380	X	29/ 5/28	19366	A	20/12/28
19357	A	3/ 1/29	19381	A	20/12/28	19368	A	3/ 1/29
19358	A	3/ 1/29	19386	A	20/12/28	19370	A	3/ 1/29
19359	A	3/ 1/29	19391	A	20/12/28	19384	A	31/ 1/29
						19385	X	30/ 5/28
						19390	A	3/ 1/29
						19392	A	3/ 1/29
						19384	A	20/12/28
						19398	X	19/ 9/28

Two of these sheep (14534 and 15699) showed a few localized lesions of jaagsiekte in only one lung when killed at the Abattoir. These two sheep were in excellent condition and revealed no symptoms of jaagsiekte whatsoever when destroyed. Some of the Onderstepoort sheep at Tweespruit died of geilsiekte and other causes, while a few were stolen, but in not a single instance were symptoms or signs of jaagsiekte detected. These sheep were daily inspected by the farm officers.

(*d*) The following experiments (S. 3490, S. 3339, and S. 3397) were undertaken to ascertain whether susceptible sheep treated in various ways can be infected by being placed in contact with infected animals:—

(i) Sheep infected with lungworm:—

14226
15977
16018
16021
16694
16699
16700

(ii) Sheep bled large quantities of blood at intervals varying from 3-7 days:—

16018
16021
16694
16700
15344
15678
15967

(iii) Sheep infected with *Strongyloides papillosis*:—

16095
16036
15931
15907
15876
16030

(iv) Splenectomized sheep:—

8429
8428
10944
10743
16023

(v) Sheep in which tar was applied to the skin at various intervals:—

10944
10743
18347
18364
18348
18001
18349
16532
16703
18353

In none of these sheep was there any evidence of jaagsiekte. With the exception of splenectomized sheep 8429, 10743, and 16023, which are still alive, all were slaughtered and the lungs carefully examined. The splenectomized sheep are in excellent condition, and not showing any symptoms.

The experiments dealing with the possibility of the transmission of jaagsiekte or some other lung trouble in sheep by exciting causes such as organisms, dust, etc., is proceeding and will be more fully reported on in a later paper.

Discussion.

About 100 sheep have been kept in various ways in contact with animals affected with jaagsiekte. Only three animals became affected with very slight localized lesions of jaagsiekte. These were of such a nature that they did not in any way interfere with the health of the sheep. In fact, these lesions would have been missed by a layman. From the above it will be seen that it was not possible by the in-contact experiments to produce clinically affected cases of jaagsiekte. De Kock (1929), in a consideration of the histo-pathology of jaagsiekte, expressed the view that the disease is probably of the nature of a neoplasm, i.e. a multiple papilliform cyst-adenoma. Moreover from the experiments conducted at Onderstepoort and at Tweespruit it can safely be said that jaagsiekte does not belong to the category of an ordinary infectious disease. It may be argued that the predisposing conditions were not present at Onderstepoort (e.g. climatic conditions, etc.), to set up the disease by close contact. The disease did not, however, develop on the lines of an infectious disease in the Onderstepoort sheep kept at Tweespruit. At Tweespruit such predisposing causes must have been present, in view of the high mortality from jaagsiekte amongst Tweespruit sheep. Mitchell maintains that he was justified in stating that under experimental conditions lesions may be found in in-contacts in from 3-5 days. As pointed out above, lesions of jaagsiekte may have been present in the sheep before the experiment commenced, seeing that these sheep were drafted from a known infected area. Unfortunately Mitchell does not describe the histo-pathology of the positive experimental cases, i.e. whether adenomatous foci were present, or whether the lesions were of the nature of a catarrhal pneumonia, etc. . . . Mitchell, however, does not regard the adenomatous proliferations as characteristic for the disease. He did not find such lesions present in all his cases.

De Kock (1929) pointed out that the lesions detected in a number of the sheep at the Graaff-Reinet Experimental Station resembled those described by Mitchell, and in which the characteristic adenomata of jaagsiekte were not identified. The writer is, therefore, of the opinion that Mitchell was probably dealing with a specific form of pneumonia in sheep, which was not the so-called jaagsiekte of Tweespruit studied by De Kock. In-contact experiments are being conducted with such Graaff-Reinet infected sheep in order to ascertain whether in South Africa there are two specific lung diseases of sheep:—

(1) Chronic catarrhal pneumonia, etc., described by Mitchell, and seen in some of the Graaff-Reinet Experiment Station sheep;

(2) multiple papilliform cyst-adenomata of the lung complicated with pneumonia, i.e. true jaagsiekte.

Most likely the progressive pneumonia of Montana, to which Cowdry refers, belongs to the former group, i.e. the specific pneumonia of sheep studied by Mitchell.

Can the view expressed by De Kock, viz., that the lesions of true jaagsiekte are of the nature of a neoplasm, be explained on etiological grounds? In referring to the literature it will be found that many investigators record the occurrence of spontaneous lung tumours in mice. Murphy and Sturm (1925), in their study of primary lung tumours in mice following cutaneous application of tar, found that mice failing to develop skin cancer are found to have tumours in the lungs. Incidentally it may be pointed out that these tumours seen by Murphy and Sturm resemble those in jaagsiekte sheep to a certain extent. Although spontaneous tumours in the mouse are frequently observed, yet these authors maintain that they rarely occur in such young animals, or in such high percentage of individuals as obtained by them experimentally.

Maud Slye (1927) and other workers are of the opinion that in mice there is an hereditary tendency to cancer. According to her, with double cancerous parentage the offspring will be 100 per cent. cancerous. She also refers to the organ susceptibility. She does not consider cancer to be contagious, because the most painstaking and long-continued experiments have never succeeded to transmit cancer by contact. She believes that there are apparently two factors necessary for the production of cancer: first the inherited susceptibility (i.e. susceptible soil), and secondly, irritation or chronic stimulation or trauma. In mice resistant by heredity no amount of irritation or trauma has ever induced cancer, and in mice susceptible by heredity to only one location of cancer no amount of irritation or stimulation applied to other parts of the body has even to date induced a neoplasm.

Jackson (1928), in dealing with certain biological aspects of cancer, refers to the opinion of Little that inheritance in animals has been proved, but we are not positive of the exact mechanism of this inheritance factor. Murray thinks that environmental changes do alter tumour incidence in mice, e.g. enforced non-breeding delays very markedly the age of tumour incidence.

In view of these data the suggestion that jaagsiekte in sheep is of the nature of a neoplasm may therefore be seriously considered for the following reasons:—

(*a*) The histo-pathology of the lesions;
(*b*) the low percentage mortality in the flocks;
(*c*) the failure to transmit clinical cases by close contact;
(*d*) acceptance of the tumour hypothesis would render intelligible the fatal outcome in all recognizable cases of the disease.

Hereditary factors are being investigated and attempts are being made to breed " more susceptible " animals from infected rams and ewes.

As pointed out above, the systematic slaughter of all sheep showing symptoms of jaagsiekte has been responsible for a reduction in the percentage mortality on badly infected farms. This may incidentally be due to the fact that infected ewes and rams are eradicated from the flocks, thus preventing sheep susceptible to this neoplasm being bred.

CONCLUSIONS.

(1) The greatest difficulty encountered in the study of jaagsiekte in sheep was the fact that it was practically impossible to diagnose the disease in its earliest stages, i.e. before clinical symptoms had manifested themselves. Sheep supposed to be healthy may not be free from jaagsiekte lesions.

(2) It would appear that young sheep and even lambs are liable to become affected.

(3) The mortality of jaagsiekte at Tweespruit, Orange Free State, in 1927 was 4 per cent., and in 1928 below $2\frac{1}{2}$ per cent. There it would appear that the hamels were more susceptible than the ewes. Probably rams are even more susceptible than hamels.

(4) At Tweespruit, in spite of the closest observation, it was impossible to say at what time of the year jaagsiekte was most prevalent. There the disease was, however, associated with an area relatively dry in summer and cold in winter.

(5) Sheep may remain alive for considerable periods (months, it is even believed for years) after they have actually shown symptoms of jaagsiekte.

(6) In view of the recent in-contact experiments and field observations, jaagsiekte should be contracted far more readily and to a much larger extent, if it is to be regarded as an infectious disease set up by mere contact. It was not possible to produce clinically affected cases of jaagsiekte by in-contact experiments.

(7) It was not possible to transmit jaagsiekte by close contact to sheep whose resistance had been lowered by lung-worm infection, bleeding large quantities of blood at certain intervals, *Strongyloides papillosis* infection, splenectomy, and application of tar on the skin.

(8) It is suggested that there may be two specific lung diseases of sheep in South Africa: (*a*) a chronic catarrhal pneumonia with fibrosis and lymphoid hyperplasias(?), described by Mitchell, and (*b*) multiple papilliform cyst-adenomata of the lung, with a terminal pneumonia.

(9) The suggestion that jaagsiekte of sheep is of the nature of a neoplasm may therefore be seriously considered for the following reasons:—

- (*a*) the histo-pathology of the lesions,
- (*b*) the low percentage mortality,
- (*c*) failure to produce clinical cases by in-contact experiments,
- (*d*) fatal outcome in all recognizable cases of jaagsiekte.

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15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

*Anatomical Studies No. 7: Hypoplasia of a Testicle and Hyperplasia of the Prostate in a Dog.

By Professor H. H. CURSON, F.R.C.V.S., Dr.Med.Vet., Division of Veterinary Services, Onderstepoort, Pretoria, South Africa.

The former condition was observed in the *left* undescended testicle (abdominal) of a young adult mongrel dog (D.O.B. 616) which died on 7/5/28 in an experiment conducted in the Department of Physiology. Cryptorchidism in the dog is an uncommon condition, and, when recorded, a histological examination of the retained testicle is generally omitted. Corner (?), in describing the minute anatomy of the testis of a hermaphrodite pig, refers to " numerous interstitial cells but without germ-cells in the tubules." Crew (1922), with more material, notices in the horse the abundance of spermatogonia and spermatocytes which appeared " to undergo degeneration, presumably of a fatty nature." In such cases the cells in certain fields were " separated from the basement membrane as a complete cast." In other fields the foetal state persisted, i.e. the seminiferous epithelium consisted of Sertoli cells and spermatogonia (Joest 1924).

In the case in question the testicle was softer and smaller than normal and was situated near the annulus inguinalis abdominalis. On microscopic examination the following changes were noted:—

The tunica albuginea was poorly developed, but the intertubular stroma was well marked, there being present groups of interstitial cells especially where several adjacent tubules approached each other. The cytoplasm of these cells, generally polygonal in shape, stained deep pink (with haemalum-eosin), whereas the nuclei, usually spherical, were deeply chromatic.

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* Cases of anatomical interest encountered in the Anatomical Department of the Faculty of Veterinary Science (Transvaal University College) are published under this title. The following are previous contributions to the series:—

No. 1. " A Rare Case of Cryptorchidism in a Monorchid Ram " (1927), *Vet. Jl.*, Vol. 83, p. 579: Quinlan, J. B., and Curson, H. H.

No. 2. " A Case of Reduction of the Thoracic Vertebrae in a Donkey " (1927), *S. Afr. Jl. Sc.*, Vol. 24, pp. 450-1: Curson, H. H., Jackson, C., and Neitz, W. O.

No. 3. " ' Gut-tie ' or Strangulatio Ducto-spermatica (Walch) in a Merino Wether " (1928), *Jl. S. Afr. Vet. Med. Assn.*, Vol 1, No. 2, p. 97: Curson, H. H., and Quinlan, J. B.

No. 4. " An Anomaly of the Renal Pelvis in a Horse " (1928), *Vet. Rec.*, Vol. 8, p. 1006: Curson, H. H.

No. 5. " Ossification of the Fascia Lata in a Horse " (1928), *Vet. Rec.*, Vol. 8, p. 1006: Curson, H. H., and Rossiter, L. W.

No. 6. " An Unusual Feature of the Musculature in the Shoulder of a Donkey " (1929), *Vet. Rec.*, Vol. 9, p. 66: W. S. B. Clapham.

The epithelium lining the seminiferous tubules varied greatly in arrangement and amount. The layers of cells numbered as a rule from 1 to 3, the nuclei being generally large and vesicular, whereas the cytoplasm, staining pale purple, seemed to have degenerated. Everywhere it was drawn out into filamentous strands which gave an irregular network appearance to the lumen of the tubules. In some places spermatogonia and spermatocytes were easily recognized, many of these cells being vacuolated. In no case was a spermatid seen. The lumen of other tubules was entirely filled with cells, many of which stained deep pink with a deeply chromatic nucleus. In some cases there was definitely a separation of the cellular elements from the membrana propria, suggesting cast formation. (See Fig. 1.)

Since cryptorchidism represents not only interference with caudal migration of the testicle, but also invariably arrest in the development of sex cells, the changes described above can be readily appreciated.

The latter of the two conditions under discussion is not uncommon in *old* subjects, being frequently associated with difficulty in micturition. Actually in Dog 616 marked retention of the urine was observed on post-mortem examination. Whether the hypertrophy of the prostate was related to the atrophy of the left testis it is impossible to say; but Ellenberger and Baum (1921) state that the size of the former gland " ist umgekehrt proportional der Grösse der Hoden." This statement, however, is hardly correct for the normal male. Since in a castrated dog there is marked atrophy of the prostate, one would expect that in senility a similar change would follow. (See Fig. 2.) Histological examination showed that the enlargement in Dog 616 was due to a uniform hyperplasia. (See Fig. 3.)

I desire to thank Mr. J. I. Quin, B.V.Sc., for handing this material to me.

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ELLENBERGER, W., AND BAUM, H. (1921). " Handbuch der Vergleichenden Anatomie der Haustiere," p. 543. (August Hirschwald, Berlin.)

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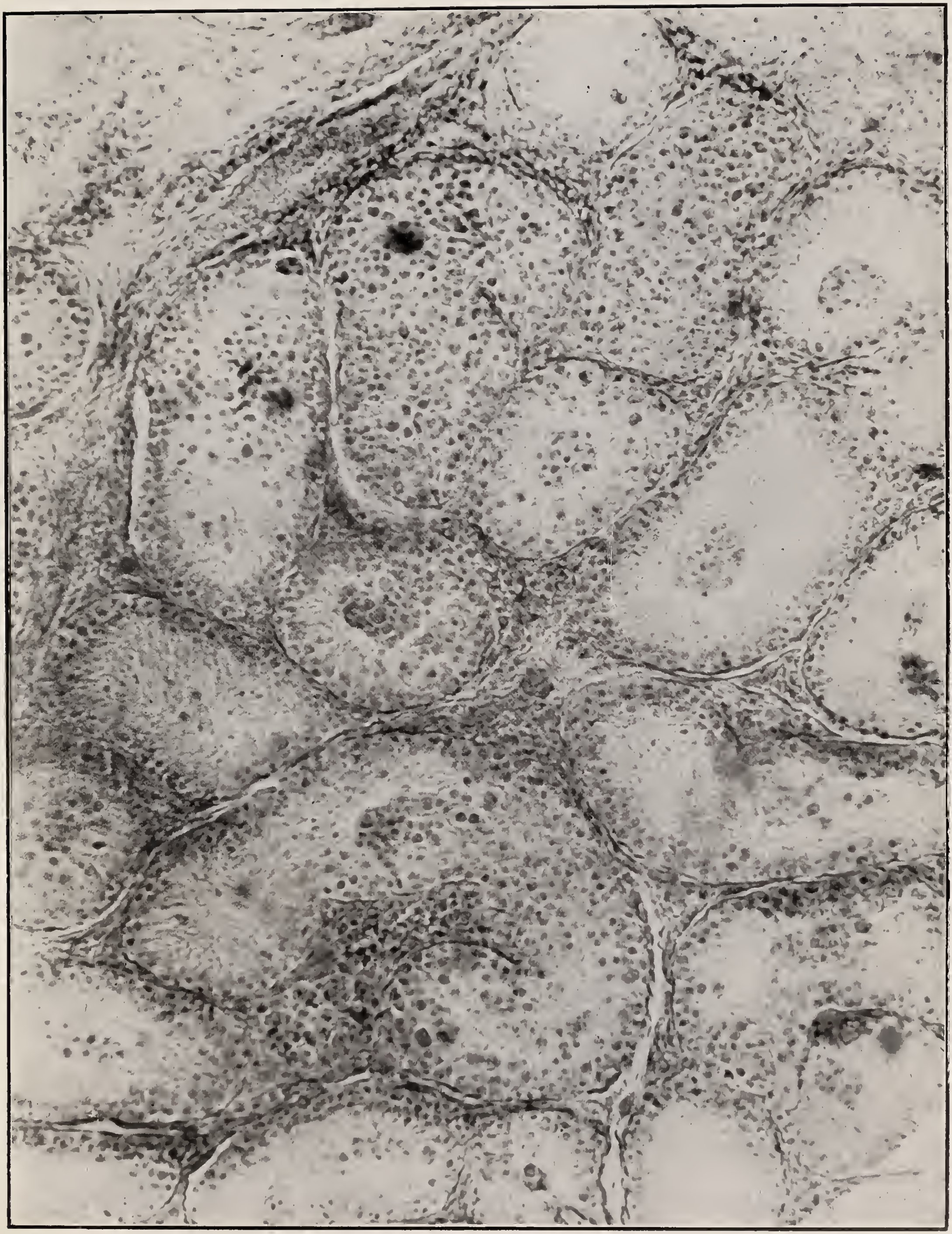
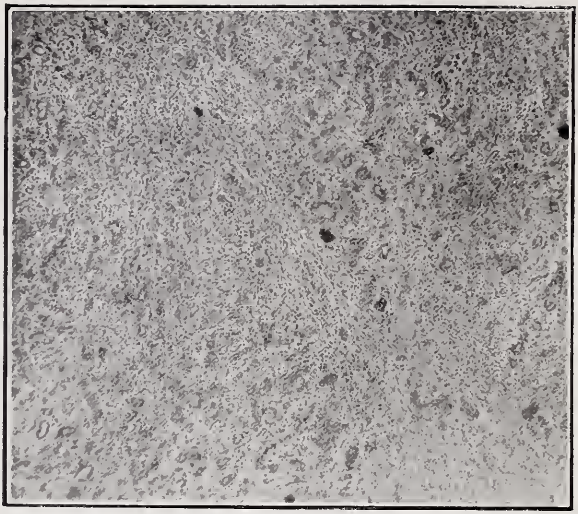
FIG. 1.



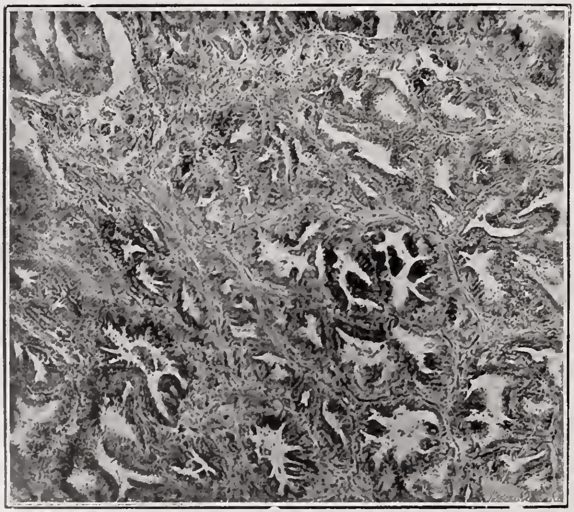
Cryptorchid testicle. Dog 616. × 160.

FIG. 2.



Section of a *castrated* dog's prostate, showing atrophy of prostatic lobules.

FIG. 3.



Section of dog's prostate (616), showing hyperplasia. × 65.

15th Annual Report of the Director of Veterinary Services,
Union of South Africa. October, 1929.

On the Toxicity of Arsenic to Fowls.

By J. P. VAN ZYL, B.A., Ph.D., Research Officer, Onderstepoort.

INTRODUCTION.

Arsenical preparations are amongst the most useful general insecticides known. In South Africa they are in almost daily use for a variety of purposes, chief amongst which might be mentioned its use as a dip against ticks or as an internal remedy against worms by stock farmers, as a spray against insect pests by horticulturists, as a bait or spray against swarming locusts by the agriculturist in in general, and finally as an exterminator of ants and vermin by the householder. Cases of suspected arsenical poisoning of animals, both accidental and malicious, are therefore fairly common, and probably run into several thousands* annually. From the standard textbooks one unfortunately gets very little definite information about the toxic dose of arsenic for the different classes of animals, or about the interpretation of analytical results (Lander, 1912).

Green of this Division undertook fairly elaborate experiments some ten years ago, with a view to determining the fate of arsenic in the animal body, the probable toxic dose for different classes of stock, and the concentration of As_2O_3 in the stomach contents, liver, etc., of animals known to have died as a result of arsenical poisoning (Green, Green and Dykmann, 1918). According to this authority " 0.1 to 0.2 mg. As_2O_3 per 100 gm. of liver tissue is difficult to interpret in a country where arsenical dipping is extensively practised; 0.4 to 0.8 mg. would be suspicious, depending upon the circumstantial evidence; above 1 mg. would be highly suspicious under most circumstances; and 2 mg. or more (per 100 gm. liver) would be an almost certain indication of fatal intoxication—could account for death irrespective of other causes " (Green, 1918). The results of analyses of stomach contents (rumenal, abomasal, etc.) were difficult of interpretation, but Green concluded that " a combination of analyses of stomach contents and liver tissue may, therefore, be easier to interpret than either alone " (Green, 1918). Subsequent experience of Green and the writer, on several thousand samples of livers and stomach contents from suspected or known arsenical poisoning cases, has in the main confirmed the figures just given.

As a continuation of Green's earlier work, and in view of the possible dangerous arsenical content of locust meal, sometimes largely used as a poultry food, it was considered advisable to obtain approximate data on the toxicity of arsenic for fowls. The details of these experiments (which were carried out in 1924 already) are here recorded.

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* At present about 800 samples are examined annually by this Laboratory in connection with arsenical poisoning.

TOXICITY OF WHITE ARSENIC.

(i) Fowl No. VII, weighing only 1.0 Kg., was given 75 mgs. of white arsenic, in the form of a very fine powder thoroughly incorporated with about 10 gm. of mealie meal, and made into pellets of a suitable size after moistening with water. The next morning the fowl was noticed to be ill and off its feed. The comb was slightly purplish and the bird was lying down most of the time. It died 44 hours after dosing. The post-mortem* revealed the chief lesion to be corrosion of the mucosa of the stomach, it being practically "burned" through. The submucosa of the stomach was very much inflamed, the ileum was covered with blood and in general the intestinal tract showed haemorrhagic enteritis. The organs all appeared normal and the tissues were not congested. The liver showed the low arsenic figure of ± 0.25 mg. As_2O_3 per 100 gm. tissue, though the stomach contents still contained a total of 6 mgs. As_2O_3, equivalent to 23 mg. per 100 gm.

The action of the arsenic in this case can, therefore, be described as local, severely irritant. The low As_2O_3 figure for the liver indicates that the arsenic was too unsoluble to be rapidly absorbed into the blood stream. We have, therefore, in this case an example where a correct diagnosis is not possible from the analysis of the liver alone, though the arsenical content of the stomach is so high as to indicate at once the likelihood of arsenical poisoning.

Conclusion.—In this case 75 mg. per Kilogram (or 7.5 gm. As_2O_3 per 100 Kg. body weight), in the form of finely powdered white arsenic administered in a food mixture to a young fowl, proved fatal within two days.

(ii) Fowl No. X, weighing 1.8 Kg, was dosed with 320 mg. of a different sample of white arsenic, mixed with a trace of fat but otherwise undiluted. Within a day the bird appeared to be dull and the comb showed slight discoloration. Before the end of the second day the fowl looked very ill and had a dark coloured comb; it was continually lying down and refused food; subsequently a mucous, offensive-smelling discharge from the beak became frequent, especially when the bird was disturbed. Death ensued four days after the administration of the arsenc. The post-mortem examination showed the immediate cause of death to have been severe necrosis of the stomach, accompanied with gangrenous decomposition of its mucosa. The submucosa of the stomach was also much inflamed. With the exception of a slight irritation of the jejunum and a barely perceptible oedema of the lungs, there were no further abnormalities. Liver, heart, kidneys and the major portion of the intestinal tract were apparently unaffected by the poison. Chemical analysis revealed only a trace of As_2O_3 (less than 0.15 mg. per 100 gm.) in the liver. In the stomach contents considerable arsenic was found to be present, but owing to an unfortunate accident, its quantitative determination was not possible.

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* Practically all the post-mortems referred to in this paper were carried out by Dr. Veglia, a veterinarian of considerable experience in arsenical poisoning cases, on the Onderstepoort staff. In a few cases the post-mortem was conducted by Mr. Martinaglia, also a veterinarian on the staff. The writer wishes to thank these colleagues here also for their valuable assistance and interest in the investigation.

In this case the action of the arsenic was even more " local " than in the first case, and death was perhaps due to its strong caustic action and not to its action as an organic poison. The difference between the two cases might have been due to the possibility of the smaller dose, diluted with meal, passing more rapidly into the intestinal tract than the larger undiluted dose, which would begin to have a corroding action on the stomach itself practically at once, but no marked action on the intestines. Here also a diagnosis of arsenical poisoning would not be possible from the analysis of the liver, though the analysis of the gizzard contents would be a much better indicator.

Conclusion.—A dose of 18 gm. As_2O_3 per 100 Kg. body weight, as ordinary white arsenic given unmixed with food, proved fatal to a fully mature bird in four days.

(iii) Fowl No. XI, weighing 2.25 Kg., received 160 mg. of the same white arsenic in a similar way to the previous fowl (No. X). After one day this bird showed no visible symptoms at all. After two days, however, it was noticed to have a slightly purplish comb, and appeared rather dull and unwell. The following day the comb and lobes were very dark and some slimy material was occasionally discharged from the beak. Nevertheless, this bird did not seem to be seriously ill, as it continued feeding and did not assume the markedly drooping attitude noticed in the first two cases. On the fifth day it was clearly beginning to improve and the last trace of sluggishness had vanished before the week was out; the comb and lobes had resumed their normal colour; the posture and gait were normal, and the appetite good. The bird was kept under observation for a further 18 days, during which period it stayed completely healthy. It was then killed and post-mortemed. All organs, except the gizzard and small intestines, seemed to be normal and healthy. The jejunum showed a faintly perceptible icterus and a slight (doubtful !) thickening, and the ileum showed traces of hyperaemia. As regards the stomach, a peculiar extension, somewhat akin to prolapsis of the stomach occasionally noticed in sheep suffering from arsenical poisoning (Theiler, 1912), was found to be present. In this part of the stomach there was an accumulation of partly decomposed, bad smelling food material. The prolapsed portion weighed in all 13 gm., of which 5 gm. were stomach wall and 8 gm. food material, yielding on analysis 8 mg. of As_2O_3. The liver showed a minute trace of arsenic, too small for exact determination by the method used (Green, 1918, and Van Zyl, 1923), but estimated at *less* than 0.04 mg. per 100 gm. tissue. The crop contents were completely free from arsenic.

The bare evidence of the chemical analysis of the portion of the stomach would tempt one to give a verdict of arsenical poisoning. In this case arsenical poisoning is, however, out of the question since the bird was actually killed in good health 23 days after dosing, so that we have a good illustration here of the errors an analyst is liable to make if he does not know all the facts of the case.

Conclusion.—A dose of 7.1 gm. As_2O_3 per 100 Kg. body weight, as ordinary white arsenic, given unmixed with food to a mature bird, produced some degree of illness, but was not fatal.

SUMMARY.

From these three cases it would appear that ordinary white arsenic (insoluble) is much less poisonous for fowls than is commonly accepted for the dog or human subject (Blyth, 1895, Glaister, 1921). Even cattle and horses seem to be rather less resistant (Fröhner, 1910, Lander, 1912). The minimum lethal dose, which very likely would vary considerably according to the individual characteristics of the bird, the fineness of the arsenic grains, the mode of administration, etc., is placed by the recorded experiments at about 7.5 gm. As_2O_3 per 100 Kg. body weight or 150 mg. ($2\frac{1}{2}$ grains) per average large bird of 2 Kg. ($4\frac{1}{2}$ lb.) live weight. This figure agrees closely with that of 0.1-0.15 gm. per bird (weight of birds not stated!) given by Corvenin (Fröhner, 1910, and Lander, 1912).

TOXICITY OF SODIUM ARSENITE.

(i) Fowl No. II, weighing 2.0 Kg., was dosed with 100 mg. As_2O_3 in the form of 80 per cent. sodium arsenite dissolved in a little water and made up with mealie meal into pellets. It showed no symptoms on the late afternoon of the day of dosing (i.e. after five hours), but was found dead on the following morning (i.e. after 19 hours). The post-mortem showed general severe catarrhal enteritis, with hyperaemic patches, slight oedema of the lungs; congestion of the liver; patchy marked hyperaemia of the submucosa of the gizzard, with slight swelling of the mucosa. Chemical analysis revealed the presence of 1.3 mg. As_2O_3 per 100 gm. of liver tissue. In the stomach contents only 1 mg. As_2O_3 (corresponding to 3.2 mg. per 100 gm.) was left, showing that the passage of the soluble arsenic into the intestines had been rapid.

The post-mortem findings, therefore, point to arsenical intoxication, apart from a mere caustic action. Both analytical figures also indicate arsenical poisoning.

Conclusion.—Arsenic in the soluble form was fatal to a full-grown fowl within one day, when administered in a food mixture at the rate of 5 gm. As_2O_3 per 100 Kg. body weight.

(ii) Fowl No. VIII, weighing 1.45 Kg. was dosed with sodium arsenite mixed with mealie meal, equivalent to 50 mg. As_2O_3. After 18 hours this fowl appeared to be still in normal health, though it showed slight diarrhoea. During the second day, it showed unmistakable signs of illness, appearing listless, losing its appetite, and showing a purplish comb. Although subsequently these symptoms became more pronounced and the bird retained the lying position most of the time, from the sixth day a definite improvement became apparent. After eight days the fowl was apparently quite normal again, remaining in perfect health for the further period of a fortnight, during which it was kept under observation.

Conclusion.—A dose of 3.4 gm. dissolved As_2O_3 per 100 Kg. live weight, given in a good mixture to a young fowl, proved non-fatal but produced definite symptoms and a fairly marked degree of illness.

(iii) Fowl No. V, weighing 1.85 Kg. was dosed in a similar way with 95 mg. As_2O_3 in the form of 80 per cent. soduim arsenite. This bird developed symptoms in about a day, gradually getting worse and dying on the fourth day. No post-mortem was held in this case. Chemical analysis of the liver and stomach contents showed the presence of 0.4-0.5 mg. As_2O_3 per 100 gm. material in both cases.

Here the analytical figures do not admit of a definite diagnosis, though they are high enough to raise the suspicion of arsenical poisoning. The other evidence available in this case, however, enables one to return a verdict of fatal arsenical intoxication.

Conclusion.—Sodium arsenite administered in a food mixture to a mature fowl in a dose of 4.1 gm. As_2O_3 per 100 Kg. body weight proved fatal on the fourth day.

SUMMARY.

These three cases seem to place the minimum lethal dose of soluble arsenic for fowls at about 3.75 gm. As_2O_3 per 100 Kg. live weight or 75 mgs. per average large bird of 2 Kg. If we compare this figure with that for white arsenic, we see that for fowls the dissolved oxide is just twice as toxic as the unsoluble oxide. It would further appear that fowls are appreciably more resistant to arsenite than sheep, horses or cattle, as for these animals Kaufmann, Green and others put the minimum lethal dose of dissolved As_2O_3 at about 1 gm. per 100 Kg. body weight (Green and Dykmann, 1918).

EFFECT OF REPEATED SMALL DOSES OF SODIUM ARSENITE.

(i) Fowl No. IV, weighing 1.35 Kg., received on alternate days for a period of one month, along with its ordinary feed of grain, small quantities of dissolved arsenic mixed with mealie meal into suitable pellets. The first four doses were only 2 mg. As_2O_3 as 80 per cent. sodium arsenite each; then followed two doses of 5 mg. each, and finally 9 doses of 10 mg. each were administered. The fowl, therefore, got 108mg. dissolved As_2O_3 in all, corresponding to 9 gm. per 100 Kg. live weight. Absolutely no ill effects due to this treatment were noticed, and in spite of the fact that the bird was confined to a small cage in the laboratory for a month, it did not lose in weight.

Conclusion.—An average mature large bird, which had received a few preliminary small doses of arsenic, easily tolerated doses corresponding to 0.75 gm., dissolved As_2O_3 per 100 Kg. body weight in its food every second day tor several weeks, without suffering any visible effects or falling off in weight.

(ii) Fowl No. III, weighing 1.4 Kg., was given small quantities of dissolved sodium arsenite, mixed with meal, five or six times a week, for a period of five weeks. At first only the equivalent of 1 mg. As_2O_3 was given, which was soon increased to 2 mg., then to 5 mg. and at the end of the second week to 8 mgs. During the last week four of the doses were raised to 10 mgs. each. For the period of the experiment the fowl got a total of 171 mg. dissolved As_2O_3, which is equivalent to over 12 gm. per 100 Kg. body weight. Two days after the experiment was stopped (the bird being quite normal still), the bird was killed (chloroformed) and post-mortemed. All the organs were found to be perfectly normal and healthy; but the mucosa of the stomach showed signs of previous slight inflammation and the intestines showed slight general diffused hyperaemia—of the icteric type in the jejunum and of the mucous type in the ileum. Samples of the contents of the gizzard and intestines, of the liver, kidneys and muscle, were subjected to chemical analysis for arsenic. In no case, however, was there sufficient arsenic present to allow of a quantitative determination. In the stomach contents, kidney and liver "traces" of arsenic were apparently still present (estimated at less than 0.04 mg. per 100 mg. material), but in the other samples it was doubtful whether even a trace was present.

It was, therefore, plain that no visible ill effects were produced by this method of dosing with arsenic. As fowls fed on locust meal containing arsenic would, in practice, usually be subjected to the action of even smaller quantities of arsenic taken into the system in a similar way, this experiment seems to indicate that there would be little danger from using poisoned locusts in moderate quantities (compare later). A slight drop in the weight of the bird from 1.4 Kg. to 1.24 Kg. was found in this experiment. We are, however, of opinion that this should be attributed rather to the prolonged close confinement and lack of exercise than to the arsenic itself; though it is also possible that the limit of safety is indicated.

Conclusion.—Repeated small doses of soluble As_2O_3, mixed with food, up to 12 or 15 mg. As_2O_3 per average large bird of 2 Kg. live weight or about 0.75 gm. per 100 Kg. given five times a week for several weeks, had no detrimental effect. Analysis also showed that the arsenic was very rapidly eliminated from the system (Lander, 1912, and Green, 1918).

(iii) Fowl No. II*a*, weighing 2.2 Kg., was dosed with arsenic in the form of dissolved sodium arsenite incorporated into a mealie meal paste in the following quantities and with the result indicated hereunder:—

(*a*) 10 mg. As_2O_3: absolutely no effect noticeable during the following two days; then
(*b*) 25 mg. As_2O_3: no effect visible during the following three days; then
(*c*) 50 mg. As_2O_3: no clear effect visible during the following four days; then
(*d*) 100 mg. As_2O_3: bird noticeably suffering on the day after dosing and died on the third day.

Within 20 hours after receiving the final dose, the fowl was showing marked symptoms, which rapidly became more pronounced. On the second day it was dull and drooping, frequently lay down, had no appetite and developed a purplish colour of the comb and a red colour of the legs and skin. It died 60 hours after the final dose. The post-mortem finding was: very marked hyperaemic and catarrhal enteritis, fatty degeneration and congestion of the liver; practically all the other organs and tissues congested, and the mucosa of the stomach apparently slightly mummified.

As presumably the previous doses had already exerted some slight action on the intestinal tract and the organs, it might be assumed that our case would correspond roughly to one in which a single dose of say 110 mg. As_2O_3 had been administered. On the other hand, a single dose of rather more than 50 mgs., say 55 mgs., would be definitely non-fatal. These quantities correspond to 100 and 50 mgs. respectively reckoned per average large bird of 2 Kg. weight and are apparently in agreement with the previously recorded result (paragraph 3), that the minimum lethal dose of sodium arsenite is about 75 mgs. dissolved oxide per bird of 2 Kg. body weight.

Conclusion.—Dosing at intervals of several days with increasing quantities of dissolved As_2O_3 was relatively safe up to 45 mg. per single dose per average large bird, but death ensued if the dose was increased to 90 mgs.

SUMMARY.

The cases discussed under this section are of interest in connection with the question of the possible injurious effect of feeding laying meal, manufactured from sprayed (i.e. poisoned) locusts to poultry. It is here shown that average large fowls can easily tolerate as much as 20 to 25 mgs. ($=\frac{1}{3}$ gr.) of the dissolved oxide thrice weekly for several successive weeks. This limit will be rarely, if ever, reached by feeding poisoned locusts or locust meal at the rates ordinarily recommended. It is further of interest to note that Corvenin reported no injurious effects for 20 mgs. *white arsenic* (i.e. the insoluble, less active form) given daily for a week, but found that a dose of 50 mgs. daily for a week proved fatal (Fröhner, 1910). If we again make the assumption that white arsenic is only half as toxic to fowls as is sodium arsenite (cp. p. 1193), Corvenin's figure for safety is 10 mg. dissolved oxide daily for a week (per bird of unstated weight?), whereas our own estimate for a prolonged period is 12 to 15 mgs. given five times weekly per average bird of 2 Kg. weight. The agreement is, therefore, very satisfactory.

THE ARSENIC CONTENT OF POISONED LOCUSTS.

As has already been indicated, the main object of this study was to obtain information that would be of assistance in dealing with the question of the possible toxicity of poisoned locusts or locust meal to poultry. This section of the investigation, therefore, had for its object the analysis of various samples of poisoned locusts, so as to obtain definite knowledge of the probable arsenic content of the average article as used in this country.

During the heavy locust infestation of 1922 and subsequent years, we sometimes had occasion to examine samples of dead locusts, suspected of having been the cause of arsenical poisoning of stock. These samples showed considerable variation in arsenic content, and as their number was not large enough to admit of generalizations, an attempt was made to procure further samples from locust officers and other sources. Unfortunately very few of the officials approached could offer us the necessary assistance, and as from about the middle of 1924 swarms of voetganger locusts became relatively scarce in the Transvaal, as a result of the vigorous locust campaign of the Department, it became nearly impossible to obtain further samples. The following selected figures are, however, we think, fairly representative of the average arsenic content of poisoned locusts, so that we feel justified in using them for the purpose of drawing our general conclusions.

(i) From the O.F.S. a case was reported, where a heifer was supposed to have died as a result of feeding on some dried out locusts, that had been killed some months previously by spraying with locust poison. For, on opening the animal, the owner found a considerable quantity of locusts in the rumen and noticed that the lining of the stomachs and the intestines had in part been badly affected. A sample of the locusts was collected in the vicinity of the spot where the animal was known to have grazed, and sent to us for analysis. The average sample was found to contain 78 mg. As_2O_3 per 100 gm. of locust material. The evidence of the post-mortem makes it practically certain that death was due to the arsenic.

If we assume that the animal's weight was 300 Kg., i.e. 660 lb., that the bulk of the arsenic in or on the locusts was easily soluble during digestion, and that, therefore, ± 3.5 gm. As_2O_3 might be sufficient to cause death, it would have been necessary for the animal to ingest 4.5 Kg., i.e. 10 lb. to produce fatal effects. We, therefore, see that even with this very high arsenic content, a relatively large bulk of the fairly light, dried out locusts would have to be eaten by the animal in the space of a day or two to cause death. As it is, however, quite possible, in our opinion, that cattle grazing on poor veld may consume 10 lb. or perhaps even 20 lb. of dried locusts in a day, we see that such a high arsenical content as 75 mgs. and more per 100 gm. must be regarded as definitely unsafe. On the basis of calculation used above ± 40 mg. As_2O_3 in 100 gm. would have to be regarded as the " border value," though it would be advisable to place the limit of safety a good deal lower still, say at 25 mg. As_2O_3 per 100 gm. of dried locusts.

It should be added that such high values for dried locusts as the above are generally due to an excessive use of the poison, and would hardly ever occur if the spraying were properly done. Moreover, animals grazing on the veld are liable to pick up dangerous amounts of arsenic at spots where the poison was spilt or some poison bait left over. In such cases death would be falsely attributed to the locusts.

(ii) Another case reported from the O.F.S. was to the effect that a cow and a calf had died after feeding on locusts that had been sprayed the previous day. In this instance the possibility of spilt poison having been the cause of death had to be ruled out, as the voetgangers had been sprayed whilst still on the other side of the wire fence. Some months later, when the locusts were quite dried out, it was reported that cattle, horses, poultry and even pigs could eat their fill of them without showing the least symptoms of ill-effects. Unfortunately samples of the fresh " dangerous " locusts were not sent. The old dry and " harmless " material was, however, sent to us for analysis. The arsenic content was found to be 40 mg. As_2O_3 per 100 gm. of dry locusts.

Our above deduction that 40 mg. As_2O_3 per 100 gh. of food material might be regarded as fairly safe, is here confirmed by actual farming experience. The explanation of the different toxicity of the fresh and dry locusts is probably twofold. In the first case the arsenic on the freshly sprayed locusts is present as highly active sodium arsenite and the bulk of it is in the form of a soluble outside layer. After several months of " weathering " it may be assumed that most of it has gone over into less soluble, and therefore less toxic forms, such as the oxide or loose combinations with organic matter. Secondly, there can be no doubt that the total amount of arsenic would be much reduced by long exposure to the elements; for the bulk of the adhering poison would be washed out by the rain or blown away by the wind. In the case under discussion, the fresh material might very likely have had an arsenic content of ±100 mgs. As_2O_3 per 100 gm.

(iii) In a case from Bechuanaland, two samples of dry flying locusts, suspected of having given rise to arsenical poisoning of a horse, were submitted to us, along with a sample of the stomach contents of the horse. Chemical analysis revealed the presence of 0.6 mg. As_2O_3 per 100 gm. in the one sample of locusts, a trace of arsenic (about 0.1 mg.) in the other, and only a " doubtful trace " of arsenic in the stomach contents.

If the two samples of locusts were representative of the material eaten by the horse, arsenical poisoning due to the locusts would be entirely out of the question. As the analysis of the stomach contents further make it extremely unlikely that this was a case of arsenical poisoning, we have here an example of the not uncommon case that the sprayed locusts are wrongly regarded as responsible for deaths due to other causes.

(iv) A quantity of fresh locusts that had been heavily sprayed at our request was collected within a day of spraying. A good average sample was analysed and it was found that the arsenic content was 75 mg. As_2O_3 per 100 gm. of air-dry material.

(v) Various other samples of locusts analysed by us gave values for As_2O_3 ranging between 0.0 mg. and 103 mg. per 100 gm. of dry material.

These figures were of particular interest in those cases where symptoms or post-mortem findings were described and where samples of relicta from animals supposed to have died of arsenical poisoning following on feeding on locusts were also sent in. Where the evidence clearly pointed to arsenical poisoning, it was found that the arsenic content of the locusts was high, or that there was a distinct possibility of the animals having picked up spilt poison. In one or two cases, where the analysis of relicta seemed to exclude arsenical poisoning, the locusts were found to contain only a moderate percentage of arsenic. Unfortunately in most cases sufficient information was not available to allow of a definite diagnosis.

(vi) It might be of interest to record here that in a few instances an attempt was made to obtain the arsenic value per 100 poisoned locusts. As the dried locusts were seldom intact they usually had to be " reconstructed," so that the figures obtained were only approximate. Flying locusts were found to weigh about 35 gm. per

100 and voetgangers about 12 gm. per 100, when thoroughly air-dried. As the field conditions usually allow of a much more thorough spraying in the case of voetgangers than of fliers, it was not surprising to find that per 100 gm. weight the former contained more arsenic than the latter. In the few cases that were examined 100 voetganger locusts contained on an average 4 mg. As_2O_3, corresponding to 33 mgs. per 100 gm., whereas 100 flying locusts contained 7 mg. As_2O_3, corresponding to 20 mg. per 100 gm. of dry locusts.

Our analysis on about half a dozen samples of poisoned locusts showed that the arsenic content per 100 locusts could vary considerably, figures ranging between 2.5 and 8 mg. As_2O_3 being obtained. The larger flying locusts usually contained more arsenic per 100 locusts than the small voetgangers, though the latter usually contained more arsenic per 100 gm. For purposes of comparison, it may be noted that Theron and Hall (1924) found up to 2 mgs. As_2O_3 per 100 locusts or 3.3 mg. per 100 gm. in an experiment where a dilute poison bait (1.10) had been used.

Summary.—The arsenic content of locusts supposed to have been killed by locust poison varied considerably. The maximum values were found to be in the neighbourhood of 100 mg. As_2O_3 per 100 gm. Such high values cannot be regarded as normal and are due to excessive spraying or spilling of poison. Freshly sprayed locusts would appear to contain more arsenic and to be more toxic than veld-dried material. Voetgangers are liable to contain more arsenic in the same weight than fliers. In many cases the arsenic content was only a few mgs. or even a fraction of a mg. per 100 mg. In the large majority of cases the arsenic content was less than 25 mg. per 100 gm.

From Theron and Hall's note one gets the impression that they place the limit of safety at only a little over 3½ mg., say at 5 mg. per 100 gm. (Theron and Hall, 1924). We feel confident, however, that a much higher arsenic content would still be quite safe. On general grounds we do not think that dry locusts containing 25 mgs. or less As_2O_3 per 100 gm. could be a source of danger to farm stock. If the arsenic content lies between 25 and 50 mgs., it would be advisable to exercise some care, especially with pigs. For the rarely occurring higher values of arsenic, the material would only be safe if fed in small quantities. Freshly sprayed locusts, which may have considerable poison loosely adhering to their bodies, should preferably always be regarded as unsafe.

Many cases of mortality amongst stock, attributed by the owners to poisoned locusts, are not even due to arsenical poisoning. Local excessive use of the poison or spilling of it, and poison residues are presumably a greater cause of danger than the locusts themselves.

TOXICITY OF POISONED LOCUSTS TO FOWLS.

Although from the observations described earlier, we were of opinion that poisoned locusts would ordinarily have no ill-effects when fed to poultry, it was decided to carry out an actual feeding test, as originally contemplated. For this purpose one of the locust officers was specially requested to provide suitable material of a high arsenic value. This was obtained by heavily spraying a swarm of flying locusts that had settled in an orchard in the evening. The following morning, i.e. before the adhering poison was lost, the dead and half-dead locusts were raked together gently and a large bagful collected. This was brought to the laboratory the same day, when the locusts were spread out on trays and dried thoroughly. For the purpose of obtaining uniform material, the dried locusts were crushed up into a coarse meal. After thorough mixing, samples were analysed from different parts of the container and the arsenic content found to be 76, 70, and 80 mg., i.e. an average of 75 m.g. As_2O_3 per 100 gm. of dried locusts. This figure was high compared with the average run of field samples but for that very reason the material was the more suitable for the purpose of our experiment.

Locusts or locust meal can certainly be regarded as a very valuable food for poultry (*Journal*, Department of Agriculture, 1923), so that the question of the safety of poisoned locusts for poultry was at the time—and may in future again become—of economic importance. In practice it does unfortunately occasionally happen that the veld-dried locusts may contain an amount of poison that must be regarded as dangerous to most classes of farm stock, but as fowls appeared to be more resistant to soluble arsenic than cattle, etc., it was anticipated that, as a poultry food, locusts showing even the maximum arsenic figure would still be relatively safe.

The first feeding experiment was planned so that individual caged birds should receive locust meal one, two, three and six times weekly, in quantities which would supply one or two minimum lethal doses per week, i.e. 75 or 150 mg. As_2O_3 per bird of 2 Kg. body weight. It was found, however, that the fowls refused to eat even the smallest ration of about 15 gm. ($\frac{1}{2}$ oz.) locust meal at a time. Efforts were made to induce them to eat a sufficient quantity by cutting down the supply of grain, "spicing" the locust meal with a little salt or making it into a mash with mealie meal or bran, or even fasting the birds for a full day. These efforts were, however, only partially successful, and the average intake after a fortnight's trial was only about 15 gm. in two days. Possibly whole dried locusts would have been eaten much better, but in that case there would have been no guarantee of a uniformly poisonous food; moreover, our whole supply of locusts had been ground up and at the time a fresh supply of suitable material was unavailable. The attempts to feed large quantities of the material to birds kept in small cages in the laboratory had therefore to be abandoned, and it was decided to keep the birds under natural conditions in outside runs. On account of the shortage of runs it was not possible, however, to try out the varying doses mentioned above simultaneously, and for the first experiment the following procedure was followed. Seven birds, all cocks in rather poor condition, were put into one run. Some of them were taken from the preliminary laboratory feeding trials and had already become accustomed to the locust meal. As the other birds, however, barely touched the locust meal, it was found necessary to cut down the ordinary grain ration considerably. After nearly a week all the birds were noticed to eat the locust meal-bran mixture supplied fairly well. During the next period of nearly a week they continued eating the whole of the locust meal (about 40 gm. daily for the seven birds), as well as an increased supply of grain. It was now thought that the experiment could be regarded as properly started (15/9/24). The locust ration was now fixed for the seven fowls at 100 gm. four times weekly. On the mornings when no locust meal was given, green lucerne was supplied. The usual, not too liberal, mixed grain ration was given during the afternoon, when all the locusts had been eaten. After five days, i.e. on 20/9/24, it was assumed that the set-back in condition, suffered by the fowls in the preliminary stage of the test, would have been more or less made good, so that from this date the weights of the birds were taken and recorded. This rate of feeding was continued for a further two weeks, the fowls all apparently eating the ground locusts well and finishing the whole of the locust ration each day. It was then increased to 75 gm. daily for the seven birds, green food and approximately the same amount of mixed grain as before, being still supplied. This was maintained for a fortnight, but as some of the fowls were then beginning to show signs of getting more food than they wanted, the locust ration was stopped for a week, after which the grain ration was reduced and the locust meal increased to 100 gms. daily. This corresponded to 100 gms. locust meal per bird per week, i.e. 75 mgs. or approximately one minimum lethal dose per week. This ration was maintained for four weeks, with one break of four days in the middle. The weights of the birds at first showed a marked increase in six of the seven cases, remaining fairly stationary during the latter part of this period. Only fowl No. 5 had made no gain and later on even decreased in weight. This caused no surprise, however, as it had been noticed for some time that this particular cock was much harassed by the others. This bird was, therefore, removed from the experiment and killed. The post-mortem showed no signs of arsenical poisoning and chemical analysis revealed only traces of As_2O_3. As it was evident that the birds were getting as much food as they could eat, the grain ration was now further reduced and the locust ration increased to 150 gm. six times a week for the six fowls. This rate was maintained for two weeks, without any ill effects whatever. Finally, as a suitable ending to this test, an attempt was made to let the fowls eat as much of the poisoned locusts as they could manage, after three days of a locust-free diet. The grain ration was, therefore, stopped and, besides a little green material, the only food these fowls now got for ten days consisted of a mixture of equal parts of locust meal, mealie meal and wheat bran. Seven hundred and fifty (750) gm. of this mixture, made up into a mash, was given every morning. The excess was removed in the evening, thoroughly dried and weighed, so as to determine approximately the amount eaten. On the assumption that the different ingredients were eaten in like amounts the quantity of locust meal consumed was calculated. During the first two or three days the mixture was not taken too well, but for the whole ten days an average of 480 gm. daily were eaten, corresponding to approximately 160 gm. of locust meal or 120 mg. As_2O_3 daily for the six fowls. The experiment was then stopped. All six birds were apparently in perfect health and had retained their weights. Five days after the last feed of locusts, two of the birds were killed and roasted, with the object of finding whether the long continued use of locust meal had imparted any unpleasant flavour to the meat. No unusual flavour could, however, be detected. The post-mortem showed trivial diffused hyperaemia in parts of the intestines, otherwise nothing unusual could be detected. Chemical analysis of the stomach contents and liver showed only a trace of As_2O_3.

For the purpose of easy review the quantities of locust meal fed during the different periods of this experiment and the weights of the fowls are given in the following table:—

Dates.	Weights of Fowls in Kg.								Weekly Rate of Locusts per Bird.	Remarks.
	1	2	3	4	5	6	7	Av.		
6/9–10/9/24.	—	—	—	—	—	—	—	—	30 gm.	Preliminary period.
–15/9/24.	—	—	—	—	—	—	—	—	40 gm.	
–4/10/24.	1·92	1·45	1·32	1·67	1·62	1·55	1·52	1·58	56 gm.	Weight of fowls on 20/9/24.
–18/10/24	2·05	1·52	1·32	1·72	1·70	1·55	1·55	1·63	75 gm.	Weight of fowls on 4/10/24.
25/10–7/11/24	2·37	1·90	2·05	2·25	(1·70)	1·95	2·10	2·10	100 gm.	Weight of fowls on 4/11/24. No locusts 19/10–24/10/24.
12/11–22/11/24	2·35	1·90	2·15	2·22	(1·57)	1·92	2·10	2·11	100 gm.	Weight of fowls on 15/11/24. No locusts 8/11–11/11/24.
–5/12/24.	—	—	—	—	—	—	—	—	150 gm.	No locusts 6/12–8/12/24.
9/12–18/12/24	2·27	1·77	2·05	2·27	—	1·95	2·22	2·09	185 gm.	Exper. stopped.

From the table it is seen that the maximum amount of locust meal consumed voluntarily by the fowls at the end of the test was nearly an ounce per bird per day, a quantity which is far in excess of the recommended ration of ½ oz. per bird three times during the week (First Rept. Comm. of Control, S.A. Central Locust Board. 1907). It is further seen that the fowls were putting on weight rapidly between 4/10/24 and 4/11/24, when they were getting a ration of poisoned locusts that supplied them daily with 8 to 10 mgs. of As_2O_3. This is in accordance with the earlier observations (paragraph 4), when a daily dose of 10 mg. As_2O_3 as sodium arsenite proved quite harmless. When the quantity of locust meal was increased to supply 20 mg. As_2O_3 per bird daily, the weights remained stationary and no harmful effects of the arsenic were evident. Whether this quantity is still some distance from the limit of absolutely safety, we were not able to test out, as the fowls refused to eat larger amounts. Possibly also the continued use of the arsenic-charged material had caused the birds to acquire increased tolerance to arsenic. (Blyth, 1895.)

SUMMARY.

A feeding experiment carried out on a group of seven cocks showed that locust meal, prepared from locusts showing a very high arsenic content, was quite harmless, even when fed in quantities approaching one ounce per bird per day. This was the maximum amount which the birds would eat, and the daily intake of arsenic then was equivalent to 20 mg. As_2O_3 per bird. During the period when the locust ration was about half of this, the birds put on considerable weight, proving that 10 mg. As_2O_3 in the ration can be easily tolerated and may be even conducive to fattening. From this it follows that dried locusts or locust meal showing the exceptional figure of 120 mg. As_2O_3 per 100 gm., would still be absolutely safe as a poultry food if used in quantities up to three ounces weekly per average mature bird of 2 Kg. body weight, i.e. up to twice the recommended rate. In fact, the danger of the arsenic in sprayed locusts to poultry seems so small that one may be tempted to ignore it altogether. Only in the case of the owner who exercised absolutely no control over the food-intake of the bird, does it seen possible, and then only in rare cases, that the bird might run the risk of arsenical poisoning due to poisoned locusts. This might for example be the case if a bird that has an exceptionally low tolerance for arsenic (minimum lethal dose, say, 60 m.g. As_2O_3 per 2 Kg. weight), has access to fresh, heavily sprayed locusts (say containing 120 mg. As_2O_3 per 10 gm. of dry material) and eats an exceptional amount of the locusts (say equivalent to more than 50 gm. or about 2 ozs. of dry matter). Neither does it seem that there is any fear of a foreign flavour being imparted to the flesh of fattening birds receiving moderate quantities of locust meal in their rations, if the locust meal is discontinued a few days before killing. The effect of locusts on the quality of the egg was not studied.

We are, therefore, of opinion that the combined evidence of the experiments described here clearly shows that the danger of feeding poisoned locusts to poultry is practically negligible.

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A Clinical Case: Spasm of the Diaphragm in a Horse.

By B. S. PARKIN, B.A., M.R.C.V.S., Veterinary Research Officer, Onderstepoort.

At 3 p.m. on the 21st March, 1929, a bay mare, 13.2 h., aged 6 years, in good condition, was presented for examination. This animal had been trucked from Belfast to Pretoria the previous day and had on the morning of the 21st been driven from Pretoria to Onderstepoort, a distance of about 7 miles. When it was trotted out for inspection at about 2.30 p.m. it was apparently normal. Shortly afterwards, however, it was noticed to be dyspnoeic.

The mare, when first seen, showed "paddling" of the limbs and "chopped" continually with the fore-limbs. A slight foamy discharge was noticed to escape from the nostrils. The muscles of the facial region were markedly contracted, the eyes were staring, the nostrils widely dilated and the jaws could not be forced open. The muscles of the remainder of the body were not markedly contracted and the animal walked quite freely. Most noticeable were the rapid respirations and a very distinct sustained thumping sound which could be heard a few paces distant and which shook the animal's body. In order to study the case more closely, the mare was moved to a stable and kept under observation.

A diagnosis of spasm of the diaphragm was made on the following symptoms:

The pulse at the external maxillary artery was just perceptible but could not be taken on account of it being obscured by the vibrations already referred to. There was salivation and the conjunctiva was cyanotic. The temperature was 104° F. The heartbeat also was difficult to detect, its rate per minute being about 76-82. Further, it appeared that the two heart sounds were followed by the "thump." On either side in the upper third of the thorax were detected on percussion symmetrical areas of dulness. Auscultation was of no assistance on account of the obscuring of lung sounds by the "thump." The rate of the respirations taken at the nostrils was 84 per minute, and the rate of the "thump" was 84 per minute. The jugular vein was not distended and showed no pulsation. No side wall impact could be felt by palpation over the heart region, i.e. if the hand was held well forward towards the shoulder. On moving the hand downwards and backwards towards the xiphoid cartilage, a marked impact and vibration could be detected. This impact and vibration could also be found on moving the hand upwards along the line of the costal arch. When the hand was moved forwards and backwards from the costal arch, the vibration decreased. Immediately behind the costal arch, which was somewhat prominent, occurred a groove about 6-8 cm. broad. Immediately behind this groove the flank at intervals projected markedly. The result was that the flank region showed a "surging motion." At 4.30 p.m. the rate of "thump" was 80, rate of respiration 80, and the pulse-rate uncertain. At 7.30 p.m. examination showed no marked change in the symptoms. At 11.30 p.m. the "thump" was not so evident, in fact there was a slight general improvement. The heart sounds now could be distinctly heard preceding the "thump," but the rate had not changed. At 9 a.m. the following day, the "thump" had ceased, the temperature was 98.2°, heart-rate 76, and respiration 50. The pulse pressure was low, the groove behind the costal arch was still present but was not so evident, facial muscular contraction was absent, but the animal was not feeding and appeared dull. At 12.30 p.m. the animal was feeding and drinking; the pulse at the external maxillary artery was 72, the pulse pressure was low, and the temperature 102° F. Trotting did not distress the animal unduly, and the areas of dulness detected by percussion had disappeared.

DISCUSSION.

It was not possible to determine the heart-rate with accuracy in early stages of the condition, but judging from the regularity with which the "thump," clearly due to the diaphragmatic spasm, followed the heart-beats the rate of the heart-beats probably corresponded to that of the spasm. It is, therefore, not possible to decide that there was no connection between the spasm and the heart. The hypothesis of the cause is advanced that the condition was due to an irritation of the diaphragmatic nerve produced by an oedema or hyperaemia of the areas of the lung in the neighbourhood of the base of the heart.

No treatment was instituted and the animal became normal within 48 hours, its heart-rate, respiration, etc., being not unduly affected by lunging.

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The Intra-Abomasal Administration of Drugs to Sheep.*

By B. S. PARKIN, B.A., M.R.C.V.S., Veterinary Research Officer, Onderstepoort.

When a drug is administered per os to sheep, its uniformity of action and efficacy are influenced to a considerable extent by the variability of time that elapses before the drug reaches the intestines, by its dilution in the forestomachs, which is especially marked if it passes directly into the rumen, and by the alteration or even destruction of the drug during its passage. In the treatment of intestinal verminosis, the above-mentioned factors are of considerable importance and naturally become more and more important the further along the intestines the habitat of the parasites lies.

The problem of the medicinal treatment of oesophagostomiasis in sheep consequently becomes an extremely difficult one. Up to the present the treatment of this condition has not been crowned with success. It was with the idea of devising a method of administration of drugs to sheep which would overcome some of the above difficulties that it was decided to carry out experiments in connection with the administration of the drug directly into the abomasum.

The objects of the experiments were, primarily, (1) to decide on the most suitable site for the injection and (2) to determine the practicability of the method, i.e. whether the injection, especially if repeated, would have any detrimental effect on the abomasum and peritoneum in particular and on the sheep in general, and, secondarily, (3) to note the dosage and the effect of the various drugs used on *Oesophagostomum columbianum*.

The method used in order to determine the site of injection was to inject, by means of a 5 c.c. hypodermic syringe, coloured solutions at various points in the angle formed by the right costal arch and the middle line of the abdomen and to slaughter and examine the sheep immediately after injection. The sheep was held by an assistant in the "sitting-up" position. It was soon noticed that it was impossible to attain a uniform position in this manner, the position and bulging of the abdomen, and consequently also, to be assumed, the variation of position of the abomasum, varying with the extent of flexion of the vertebral column. Subsequently the sheep was held by the assistant in such a manner that its hindquarters were just off the ground. This was conveniently carried out by the assistant grasping the sheep in the axillae. The result was that the abdominal wall was made tense, the vertebral column always assumed the same position, and the abomasum presumably took up the same position each time in any particular animal. It was also determined that if the rumen was comparatively empty, the abomasum moved over towards the left side, and if the rumen was abnormally distended the abomasum was forced over towards the right costal arch. The success of the injection also depended on the extent of distension of the abomasum, i.e. the omount of ingesta present. The site ultimately decided on was one midway between the 8th intercostal space at its chondral aspect and the median line of the abdomen directly opposite the 8th interspace (see photograph). The direction of passage of the needle was at right angles to the abdominal wall.

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* Commenced at the Elsenburg School of Agriculture (June, 1926) and continued at the Stellenbosch-Elsenburg College of Agriculture.

Appended are the experiments in the application of this intra-abomasal method of administration. Prior to each administration faecal cultures were made on three occasions at intervals of one day to determine the presence or absence of *Oesophagostomum columbianum;* and again, commencing on the 6th day after administration, to determine whether the drug used had any effect on the parasites. If the administration completely destroyed the parasites, this destruction could be used to indicate whether the drug entered the abomasum or not. As subsequently was shown in the experiments, this indication was not of much assistance for the purpose. Consequently chief dependence was placed on autopsies when the sheep died soon after the administration. The number of sheep used in the controlled experiments was nine, one of which was lost to the experiment on account of being stolen. The controlled experiments extended over a period of one year.

SUMMARY OF CONTROLLED EXPERIMENTS.

The sheep tolerated the injections well. This is brought out by Sheep No. 34, which received, during a period of one year, ten injections, and on autopsy, one month after the last injection, showed nothing unusual. Sheep Nos. 37 and 39 were also autopsied at the same time. In both there was a gelatinous infiltration of the abdominal and abomasal walls with adhesions connecting them. This was apparently due to the entry of some of the drugs into the peritoneal cavity. Sheep Nos. 32 and 39 showed a complete destruction of *Oesophagostomum.* In sheep Nos. 32, 33, 36, 38, and 40, the autopsies showed that the drugs had entered the abomasum.

The detrimental effect of the larger doses of the Government Wireworm Remedy and the copper sulphate appeared to be due to their caustic action. Larger doses of these probably could be given if administered in weak solutions. No purgative effect could be ascribed to arecoline hydrobromide in 1-grain doses. Other drugs used produced no marked effects except at times uneasiness and distress, which, it would appear, occurred only when the drug entered the peritoneal cavity, for in five cases (Nos. 32, 33, 36, 38, and 40) no symptoms were shown immediately after the administration, even though the animals died within a few hours of the injection. In these autopsy showed that the drugs had entered the abomasum.

CONCLUSIONS.

(1) The site selected for the administration of drugs by the intra-abomasal method is at a point midway between the 8th intercostal space at its chondral aspect and the median line of the abdomen directly opposite the 8th interspace.

(2) The administration of drugs by the intra-abomasal method is possible, but cannot, at the present stage, be regarded as being a method applicable to general use.

(3) The chief advantage is that the drug can be administered in a concentrated form and delivered nearer the habitat of the intestinal parasites.

(4) The actual administration is not detrimental to the sheep and does not produce any after-effects of objectionable nature.

(5) A certain amount of information is obtained in connection with dosage and action of certain drugs.

(6) The chief objection is the varying position of the abomasum. Further work may result in the discovery of a more suitable site for injection.

DETAILS OF CONTROLLED EXPERIMENTS.

Exp. No.	Sheep No.	Sheep Weight. lb.	Drug.	Dose of Drug.	Effect on *Oesophagostomum columbianum*.	Remarks.
1	37	51	G.W.W.*	·02 gm.	—	No symptoms shown.
2	34	58	,,	·04 gm.	—	No symptoms shown.
3	32	58	,,	·25 gm.	+	Uneasiness about 15 min. after injection. Diarrhoea, oedema of lower abdominal wall, death in 50 hours, haemorrhagic abomasitis and enteritis, abomasal ulcerations. No oesophagostomes were found.
4	33	54	,,	·5 gm.	—	Uneasiness about 15 min. after injection. No diarrhoea, oedema of lower abdominal wall, death in about 12 hours, abomasal ulcerations marked. Oesophagostomes found.
5	34	58	Copper sulphate	·25 gm.	—	No symptoms shown.
6	35	41	,,	·5 gm.	—	No symptoms shown.
7	38	59	,,	·5 gm.	—	No symptoms shown immediately after injection, death in about 12 hours, diffuse hyperaemia of mucosa of abomasum and of first portion of duodenum, large chronic abscess in connection with and adhesions of large intestines. Oesophagostomes found.

* G.W.W.—Government Wireworm Remedy.

DETAILS OF CONTROLLED EXPERIMENTS—(*continued*).

Exp. No.	Sheep No.	Sheep Weight.	Drug.	Dose of Drug.	Effect on *Oesophagostomum columbianum.*	Remarks.
8	36	52	,,	1 gm.	—	Slight uneasiness soon after injection, death in 6 hours, marked extensive ulceration of abomasum-mucosa at site of injection, chronic pneumonia. Oesophagostomes found.
9	34	60	Carbon tetrachloride	·5 c.c.	—	No symptoms shown.
10	34	56	,,	·75 c.c.	—	No symptoms shown.
11	34	56	Carbon tetrachloride Chloroform	·75 c.c. 1 c.c.	—	No symptoms shown.
12	35	41	Carbon tetrachloride	·5 c.c.	—	Considerable uneasiness, which passed off in 30 min.
13	35	41	,,	·5 c.c.	—	No symptoms shown.
			Ether	·5 c.c.	—	Sheep stolen.
14	39	70	Carbon tetrachloride Chloroform Arecoline hydrobromide	1 c.c. 2 c.c. 1 gr.	—	Showed great uneasiness, which passed off in 20 mins.
15	39	70	,,	,,	—	No symptoms shown.
16	40	60	,,	,,	—	No symptoms shown.
17	40	60	,,	,,	—	Slight uneasiness.
18	34	56	Ol. chenopodii	1 c.c.	—	No symptoms shown.
19	39	70	,,	2 c.c.	—	No symptoms shown.
20	34	56	,,	3 c.c.	—	No symptoms shown.
21	40	60	,,	6 c.c.	—	No symptoms shown immediately after injection, death in 10 hours, autopsy showed a hyperaemia of the whole of the small intestine.
22	34	58	Thymol	1·5 gm.	—	No symptoms shown.
23	34	58	,,	1·5 gm.	—	No symptoms shown.
24	39	70	,,	3 gm.	—	No symptoms shown.
25	34	58	Tetrachlorethylene	2 c.c.	—	No symptoms shown. Sheep autopsied 48 hours later. Oesophagostomes were found.
26	39	70	,,	5 c.c.	+	No symptoms shown. Sheep was autopsied 48 hours later. No oesophagostomes were found. Cultural examination was not made.